TO STUDY THE EFFECT OF OXYGEN SUPPLEMENTATION IN TOURNIQUET USED LIMB SURGERIES BY USING BLOOD GAS ANALYSIS

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

APRIL 2015

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"TO STUDY THE EFFECT OF OXYGEN SUPPLEMENTATION IN TOURNIQUET USED LIMB SURGERIES BY USING BLOOD GAS ANALYSIS"** is a bonafide record work done by **Dr. SIVABALAN.R.G** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of University regulation for MD, Branch X -Anaesthesiology

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CERTIFICATE FROM THE GUIDE

This is to certify that this dissertation entitled **"TO STUDY THE EFFECT OF OXYGEN SUPPLEMENTATION IN TOURNIQUET USED LIMB SURGERIES BY USING BLOOD GAS ANALYSIS"** is a bonafide and genuine research work done by **Dr. SIVABALAN.R.G.** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University, in partial fulfilment of the requirement for the degree of MD in Anaesthesiology.

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DECLARATION

I **Dr. SIVABALAN. R.G.,** solemnly declare that this dissertation entitled **"TO STUDY THE EFFECT OF OXYGEN SUPPLEMENTATION IN TOURNIQUET USED LIMB SURGERIES BY USING BLOOD GAS ANALYSIS"** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other University or board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of Doctor of Medicine degree Branch –X (Anesthesiology) to be held in April 2015.

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TABLE OF CONTENTS

TO STUDY THE EFFECT OF OXYGEN SUPPLEMENTATION IN REDUCING THE ANAEROBIC METABOLISM IN SURGERIES DONE UNDER TOURNIQUET.

ABSTRACT

BACKGROUND:

Tourniquet usage can provide a bloodless surgical field. But it has the disadvantage of increasing the anaerobic metabolism in the non perfused limb. Oxygen supplementation before its application can reduce its anaerobic impact.

OBJECTIVE:

To study the effect of oxygen supplementation in surgeries done under tourniquet.

METHOD:

A prospective randomised case control study was undertaken in 60 ASA 1 & 2 patients of both the sexes in the age group of 18-45 years who undergo surgery using tourniquet. Preop ABG was taken to compare their baseline values .Case group was preoxygenated with 100% O2 for 5 mins followed by tourniquet application. Control group was not preoxygenated and tourniquet was applied. Post op VBG was taken in both the groups and compared using Chi Square T test.

RESULTS

The mean post op PH of the study group was 7.37±0.03 while that of the control group was 7.29±0.01 (P value <0.0001).Similarly the mean Lactate levels of the study group was 0.79±0.18 and that of the control group was 1.6±0.48(P value <0.0001).

CONCLUSION:

This study shows that preoxygenation improves dissolved oxygen thereby helps to decrease the anaerobic metabolites that arise due to tourniquet application.

KEYWORDS

Tourniquet, Lactate, anaerobic metabolism.

INTRODUCTION

Using a tourniquet to produce a blood free surgical field is accepted by everyone in surgery. There is a need for information about the systemic and local effects of tourniquet use in persons of good physical status. For a proper functional state, the peripheral tissues depend on an adequate supply of oxygen and an adequate microcirculation. Tourniquet application causes increase in systemic blood pressure, central venous pressure and heart rate. When we release the tourniquet reactive anaerobic metabolites are released into the circulation which cause vasodilatation in the capillaries of the muscles. When we apply tourniquet the temperature of that particular limb falls due to absent blood supply and when it is released this cold blood enters into the general circulation .The core temperature as a result can reduce to 0.6 degrees. Applying tourniquet for more than 30mins causes increase in acidosis, hypercapnia, increased serum potassium and toxic metabolites.

Aim of this study is to highlight the effect of oxygen supplementation in tourniquet used limb surgeries.

AIM OF THE STUDY

TO STUDY THE EFFECTS OF OXYGEN SUPPLEMENTATION ON THE METABOLIC AND ANAEROBIC CHANGES CAUSED BY TOURNIQUET.

PHYSIOLOGY OF OXYGEN TRANSPORT

Breathing results in entry of oxygen from the atmosphere to the alveoli. Carbondioxide produced by all cells of the body is brought to the lungs by pulmonary circulation and excreted via the alveoli.

Myoglobin.

It is the iron containing oxygen carrying pigment present in the muscle.Similar to haemoglobin which is found in the blood especially in the red blood cells, myoglobin helps in the transport of oxygen in the muscles.In studies conducted in animals it has been found that diving mammals contain abundant myoglobin enabling them to hold their breath for a longer period.It is found in the skeletal muscles but absent in the smooth muscles.

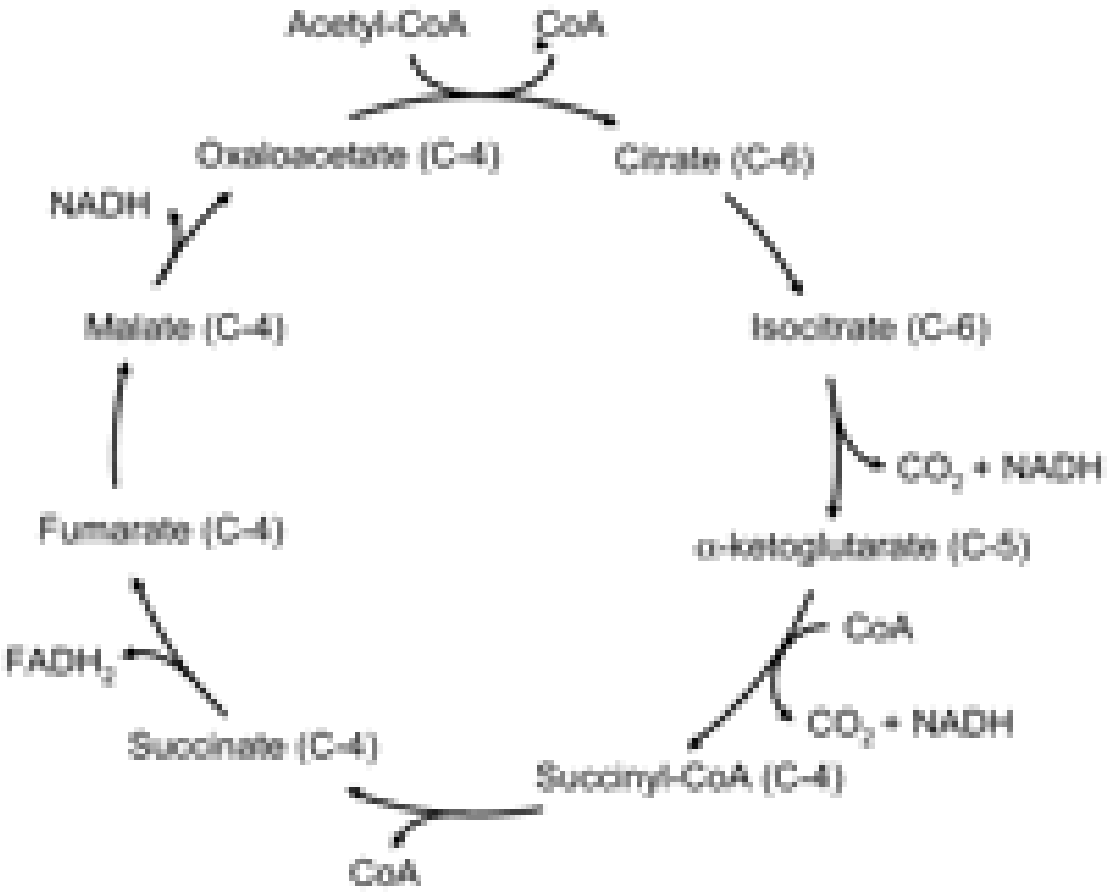
John kendrew who was awarded the nobel prize for his work on myoglobin found out that mice who were genetically engineered showed a lack of this myoglobin pigment and they also showed a reduction in the stroke volume of about 30% due to impaired myocardial contractility probably due to their defeciency of myoglobin.He has found out that these mice adapted to this defeciency by vasodilatation and reflex reactions to hypoxia.

Muscle injury and damage causes release of this myoglobin pigment in the blood stream due to rabdomyolysis enabling it to be found only in pathological states of injury.But this is nephrotoxic and has the propensity to

cause acute renal failure.Several prototypes of Myoglobin are there each serving as a potential marker for muscle injury,myocardial damage, etc. However these markers are non specific.

Cellular oxygen utilization.

The mitochondia are the primary and ultimate site for oxygen utilisation.The oxygen which we breath are taken down to the cellular level through haemoglobin which lack mitochondria.Here this oxygen enters the electron transport chain where oxygen is the final acceptor of electrons.Mitochondria goes for hibernation in cases of oxygen deprivation a term called MITOCHONDRIAL HIBERNATION.This hibernation is a low metabolic state for the mitochondria where it gains its function and complete cellular recovery once oxygen flow is re established.This mitochondrial dysfunction and hibernation are the potential factors in Multi organ dysfunction syndrome.



The electron transport chain

Airway Anatomy

Anatomically Respiratory tract in human consists of upper airway and lower airway. The upper airway includes mouth ,nasal cavities, paranasal sinuses, pharynx and larynx. Lower airway includes trachea, bronchi, bronchioles, alveolar ducts, alveolar sacs and alveoli. The distinction between upper and lower airway is the vocal cord level/CRICOID CARTILAGE.

NOSE: consists of lateral wall, floor, roof and septum. Lateral wall has turbinates, meatuses, openings of sinuses.

Blood supply is anterior and posterior ethmoidal artery (from ophthalmic artery), sphenopalatine artery(maxillary artey) superior labial artey (facial artery). antero inferior part is the zone of epistaxis called little or kasslbachs area. Venous drainage is through ophthalmic veins, maxillary vein and facial vein. **Nerve supply** of septum is septal branch of anterior ethmoidal nerve, medical posterior superior nasal nerve and nasopalatine nerve supply of lateral wall is septal branch of anterior ethmoidal nerve lateral posterior superior nasal nerve nasopalatine nerve greater palatine and lesser palatine nerves.

PHARYNX: has nasopharynx, oropharynx and laryngopharynx

LARYNX: lies opposite to C5 TO C6 vertebra. Consists of laryngeal cartilages, laryngeal ligaments, true and false vocal cords intrinsic and extrinsic muscles. Blood supply - superior laryngeal and inferior laryngeal artery branches of superior and inferior thyroid respectively.

Lymphatic drainage: supraglottic region- upper deep cervical nodes .infra glottis - lower deep cervical nodes **.Nerve supply**: external laryngeal nerve supplies cricothyroid muscle, recurrent laryngeal nerve supplies other muscles and sensory supply below the level of vocal cord. Above the level of vocal cord is supplied by internal laryngeal nerve.

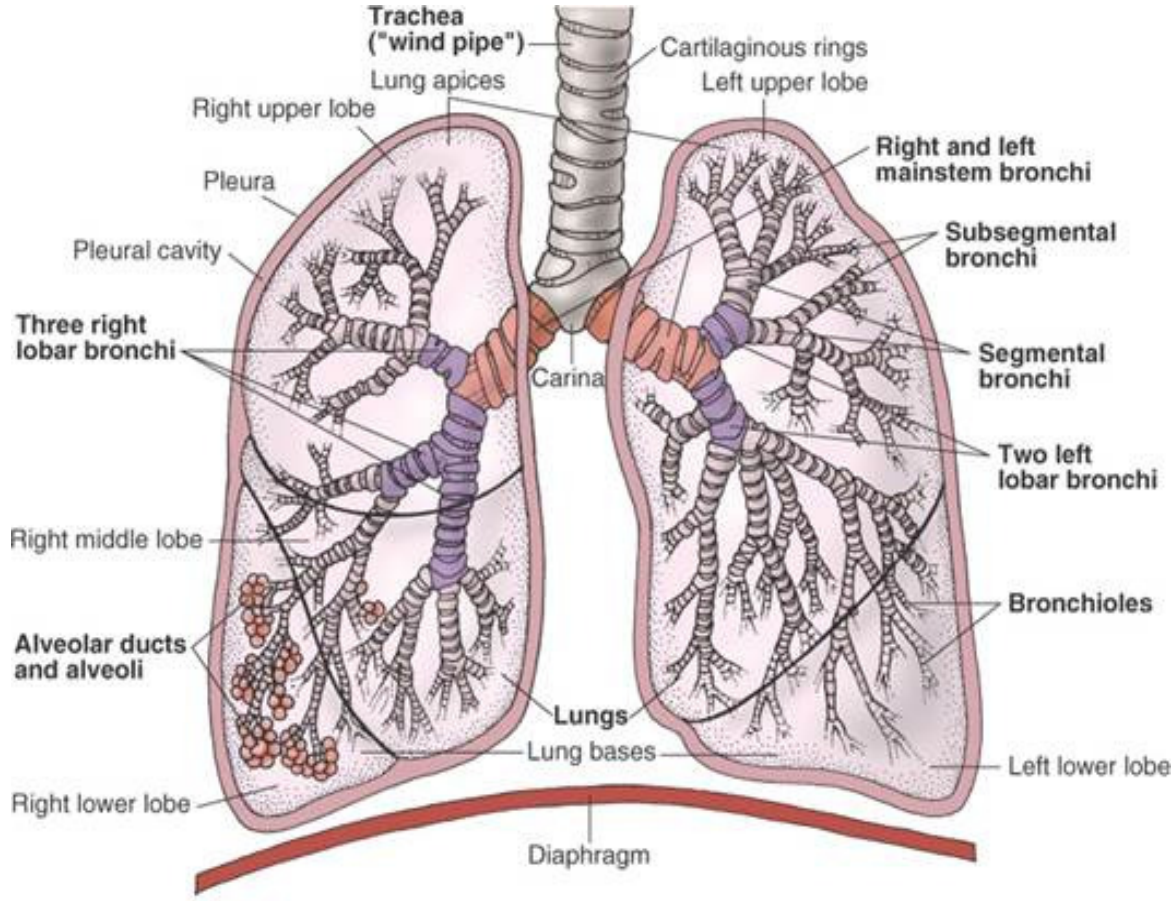
TRACHEA: largest tube in the airway.it has c shaped hyaline cartilage. It starts from larynx at C6 level and divides into two main bronchi at T4 .Bronchi and bronchioles have complete rings in the cartilage.

TRACHEOBRONCHEAL TREE

The bronchi divides progressively into 23 divisions/ generations known as bronchial tree which starts from primary bronchi to terminal bronchiole.The tracheal branching is in the form of dichotomous branching. It includes trachea, main bronchus ,segmental bronchus ,conducting bronchiole ,terminal bronchiole, respiratory bronchiole, alveolar duct ,alveolar sac and alveoli. Gas exchange takes place from respiratory bronchiole. Above this are the conducting zone generation 16 is terminal bronchiole 17, 18, 19 respiratory bronchiole 20, 21, 22 alveolar ducts 23 alveolar sacs.

LUNGS:largest part of lower respiratory tract. Enclosed within the pleural cavity .It is lined by visceral and parietal pleura. Right lung is larger than the left lung. Each lung has lobes. Right upper middle lower lobes. Left has upper lingula and lower lobe. Each lobe is further divided into segments .Totally 10 segments on each side called bronchopulmonary segments.

HISTOLOGY: Lungs lined by respiratory epithelium which changes into cuboidal epithelium down the bronchioles.It consists of cilia goblet cells glands elastin smooth muscles and cartilage.



Gas Exchange Airway:

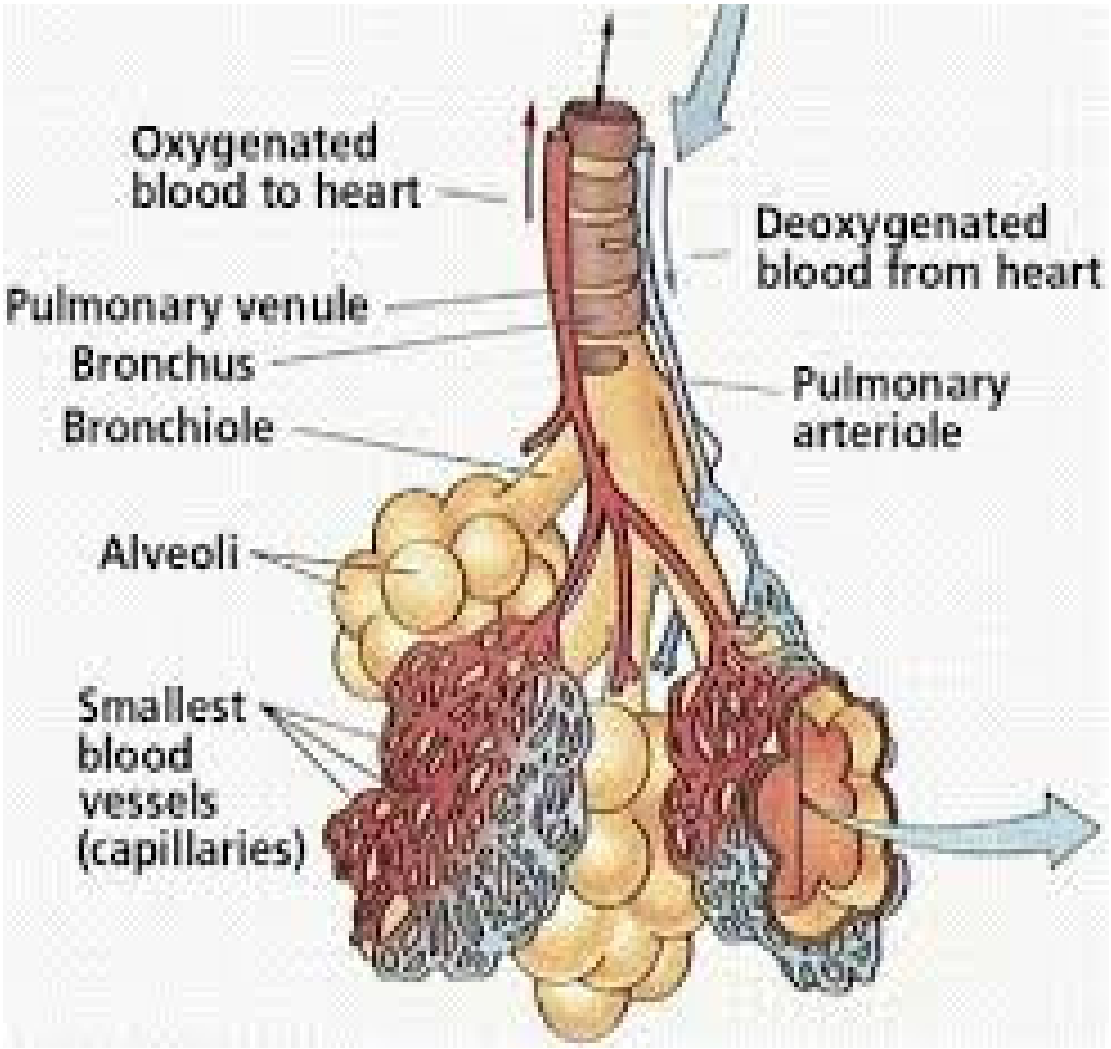
The main function of lungs is respiratory gas (O2 and CO2) exchange. Not all the atmospheric gases takes part in gas exchange Similarly not all the respiratory tract takes part in gas exchange. Thus we have 1.Conducting zone- conducts the inspired gas to the respiratory zone 2. Respiratory zone - where the exchange of gases takes place. This respiratory zone starts from respiratory bronchiole and the main respiratory organ is alveoli. Cross sectional area becomes progressively increases as we move downwards conducting zone to transitional zone thus forward velocity becomes small from the level of respiratory bronchioles so diffusion becomes the main mode of ventilation(especially in alveoli).

Other parts also take part in gas exchange to a smaller extent. But during exercise and increased production of CO2 and O2 demand exchange increases in other regions also.

Factors affecting exchange of gases are partial pressure of respiratory gases in the atmosphere, inspired gas and alveoli, blood solubility, surface area, alveolar capillary membrane barrier and its thickness blood flow across the alveoli. The main factor determining the diffusion of gases is concentration gradient across the alveolar capillary membrane and perfusion. Thus there is a need for constant supply of O2 and removal CO2 from the alveoli to maintain the concentration gradient.

Average adult male contains 300million alveoli approximately and the size of each alveoli is in the range of 75micron to 300 micron in diameter .Alveolar region volume is about 3litres. Anatomical dead space is 150ml

Physiological dead space = anatomical dead space – alveolar dead space, almost always pathological.


Oxygenated blood to heart
Deoxygenated blood from heart
Pulmonary venule
Bronchus
Bronchiole
Pulmonary arteriole
Alveoli
Smallest blood vessels (capillaries)

MECHANICS OF VENTILATION AND BREATHING.

Two phases of respiration are 1.inspiration 2.expiration

Inspiration is an active process and expiration is a passive process.

INSPIRATION

Main muscles of respiration are diaphragm push the abdominal contents downwards, External intercostals muscles pulls the rib upward and outward thus helping in increasing the lung volume. Diaphragm is innervated by phrenic nerve. Accessory muscles of inspiration are abdominal muscles, scalene muscles, sternomastoid, alar nasi. They work during exercise and increase in airway resistance.

EXPIRATION

During quite breathing expiration is passive process due to elastic recoil of lungs. But needs assistance from the abdominal muscles and internal intercostals during forceful expiration and hyperventilation, also when coughing, vomiting, defecation. The internal intercostals muscles pulls the ribs downward and inward, while the abdominal muscle

increases the intra-abdominal pressure and pushes the diaphragm upwards .All these together decreases the lung volume.

Surfactant:

Stability of alveoli depends on the surface tension and is maintained by surfactant produced by type2 alveolar epithelium. Surfactant loss leads to decreased compliance, alveolar atelectasis and pulmonary edema. Total Compliance of the lung (1/ct) =1 /compliance of lung (cl)+1/compliance of chest wall (cw).

There are regional variations in ventilation on moving down is due to the weight of the lung .The intra pleural pressure becomes less negative at the base compared to apex, also it is compressed in the resting state so expands well during inspiration.

Control of breathing:

Input/sensors –chemoreceptors, lung and other receptors.

Central control –pons medulla and other parts of brain

Output –respiratory muscles through phrenic nerves which also sends negative feedback to sensors.

Respiratory centre helps in rythmic nature of inspiration and expiration. Three main centers are medullary centre located beneath the floor of the fourth ventricle dorsal part has inspiratory neurons and ventral part has expiratory neurons apneustic centre located in lower pons. Impulses from this centre prolongs the ramp pattern of inspiration pnuemotaxic centre regulates the inspiration, thus the inspiratory volume and respiratory rate.

Sensors: Central chemoreceptors:

Located in the ventral surface of medulla .It is bathed in csf, so changes in csf and ecf pH affects the respiration. H+ ions are the powerful stimulant of respiration .But it is not easily diffusible .Its concentration increases when co2 concentration increases. Thus co2 conc in blood plays an important role in respiration.

2. **PERIPHERAL CHEMORECEPTORS**:

carotid bodies located at the bifurcation of carotid and aortic bodies located at the arch of aorta. They respond changes in PO2, PCO2 and PH. These changes are well observed in changes in arterial blood .They are more sensitive to changes in PO2 when arterial PO2 goes below 500mmHg.less sensitive to PCO2 and PH changes.

OUTPUT: respiratory muscles are described already.

RECEPTORS OF LUNG

1. Pulmonary stretch receptors plays a role in Hering breur reflex

2. Irritant receptors respond to noxious stimuli

3. J receptors responds to interstitial fluid and pulmonary capillary circulation plays a role in heart failure.

4. Other receptors:

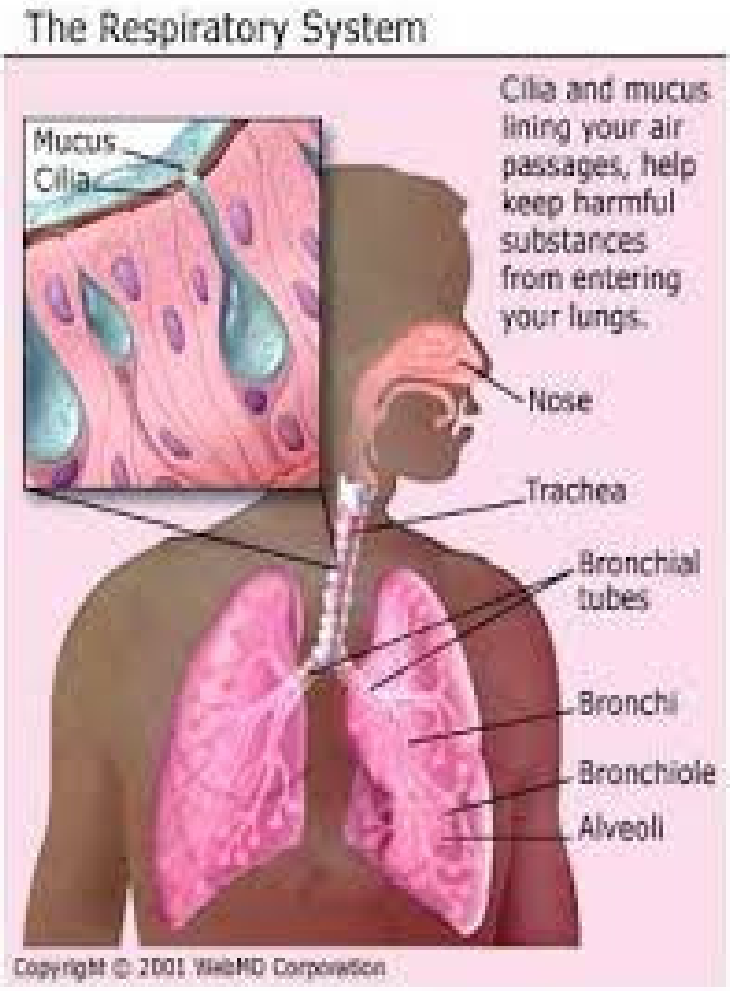
Joint and muscle receptors

Nose and upper airway receptors.

Arterial baroreceptors .

Gamma system.

Bronchial c fibres.


The Respiratory System
Cilia and mucus lining your air passages, help keep harmful substances from entering your lungs.
Mucus
Cilia
Nose
Trachea
Bronchial tubes
Bronchi
Bronchiole
Alveoli
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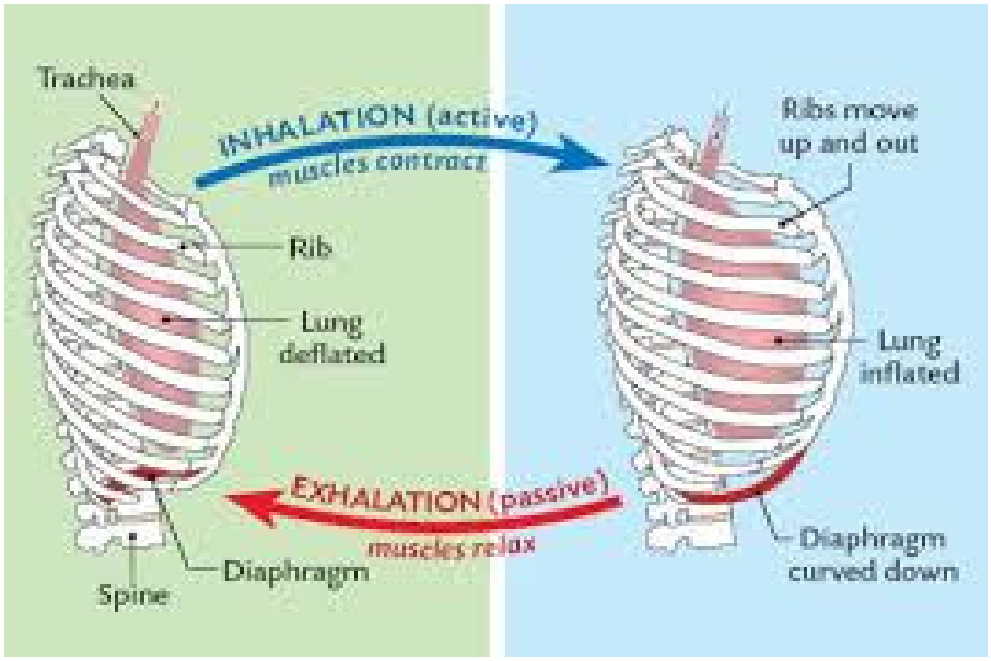

Trachea
INHALATION (active)
muscles contract
Rib
Lung deflated
Spine
Diaphragm
Ribs move up and out
Lung inflated
EXHALATION (passive)
muscles relax
Diaphragm curved down

Pulmonary Circulation:

It starts at the pulmonary artery which receives the deoxygenated blood from right heart which is rich in CO2 and other metabolites. The pulmonary artery divides into right and left pulmonary artery which finally ends in pulmonary capillaries. The pulmonary capillaries surrounds the alveoli thus the CO2 is removed and O2 is added here. This blood is carried to the left heart through pulmonary veins which is rich in oxygen. Any increase in vascular resistance at any level will affect the alveolar blood flow thus the gas exchange across the alveoli. Bronchial arteries and bronchial veins don't take part in pulmonary circulation.

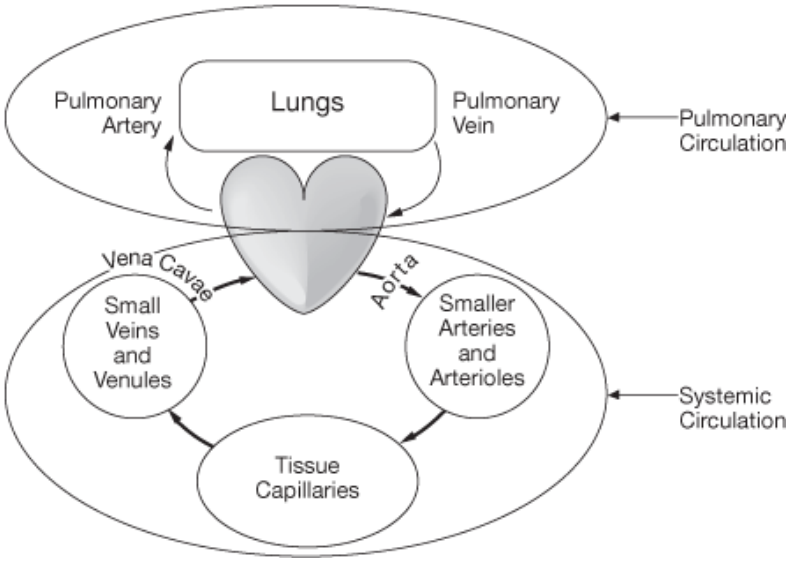
The pulmonary blood flow is not uniformly distributed. The main reason for this uneven distribution is gravity. Pulmonary blood flow is more in the dependent part in supine position whereas it is high in the base in the sitting /upright position. We have already discussed ventilation is more in the apex than base thus there is a V/Q mismatch. But studies show that prone ventilation there is better uniform distribution of pulmonary blood so better oxygenation. Hypoxic pulmonary vasoconstriction (HPV) occurs in lungs probably due to the direct effect of low PO2 .

Functions of pulmonary circulation are

1.gas exchange

2.filtering mechanism

3.converting active metabolites example angiotensin 1 to angiotensin 2.



Pressures in the Lung

It is nothing but pressure within the pulmonary circulation.It is low compared to systemic circulation.in pulmonary artery the pressure is about 25/8mmHg, mean is about 15mmHg.

Pulmonary artery -25/8 mmHg

Pulmonary arteriole-12 mmHg

Pulmonary veins-8 mmHg

Left Atrium- 5 mmHg

Left Ventricle- 120/0 mmHg

AORTA -120/80 mmHg

Systemic arteriole- 30 mmHg

Systemic capillaries- 20 mmHg

Systemic veins- 10 mmHg

Right Atrium- 2 mmHg

Right Ventricle- 25/0 mmHg

Pressure in the aorta is 60 times more than that of systemic circulation. This difference is because it supplies blood to far distant organs. But pulmonary vascular bed is close to the heart it receives whole of the cardiac output .It directs the blood from one region of lung to another during hypoxia due to hypoxic vasoconstriction.

Similarly pressure within the capillary bed varies in the lung because of hydrostatic effects i.e. pressure in the alveoli .high alveolar pressure when it crosses the capillary pressure the capillaries collapse the pressure difference between the two is called the transmural pressure which determines the capillary pressure.

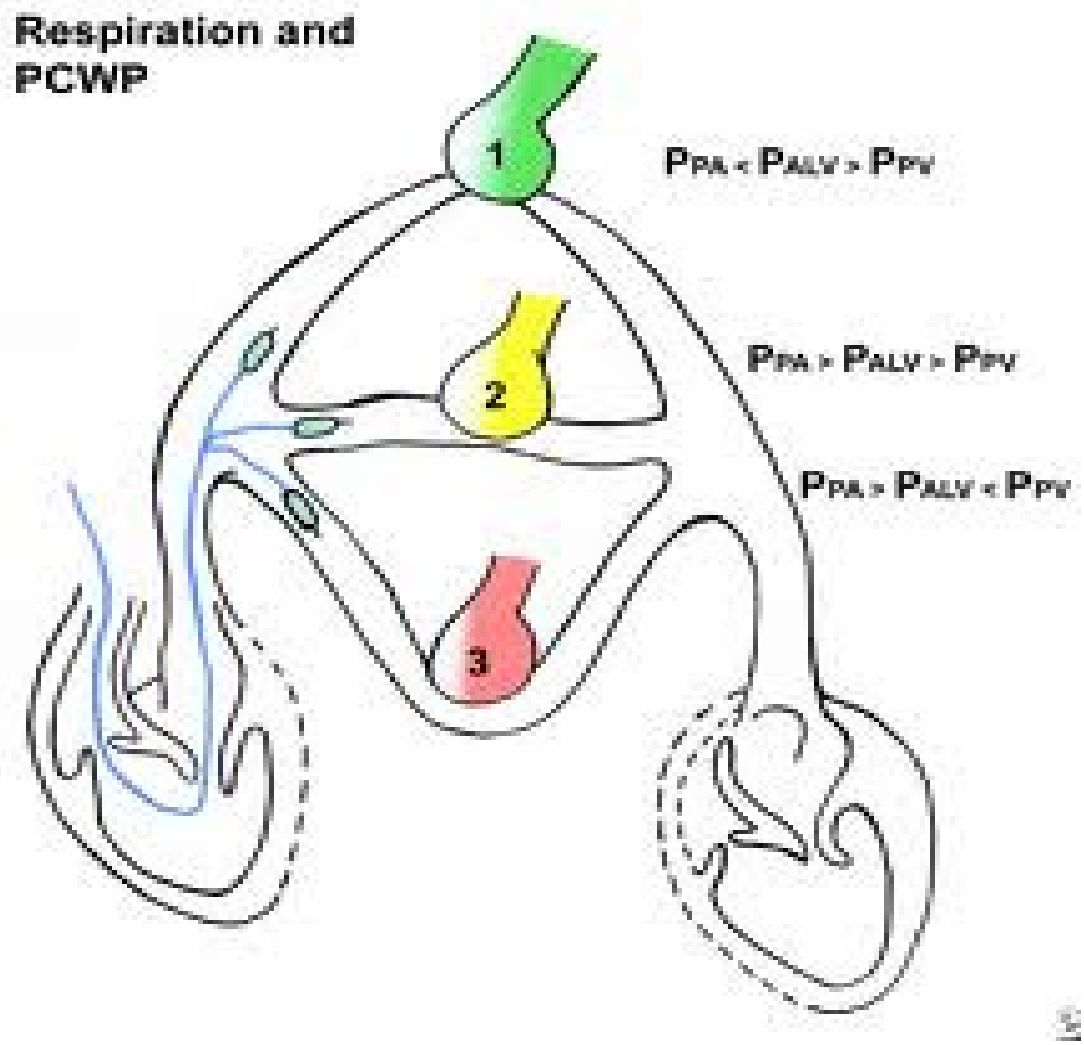
The extra alveolar capillaries are subjected intra plueral pressure around lung parenchyma. This pressure causes radial traction of the blood vessel thus the effective pressure is less compared to alveolar pressure. So both artery and vein increase in calibre when the lung expands. But capillaries surrounding the alveoli are exposed to high pressure so tend to collapse.

Lung zones are

Zone1. no blood flow;

Zone2. intermittent blood flow;

Zone3.blood flow present always.


Respiration and
PCWP
1
2
3
PPA < PALV > PPV
PPA > PALV > PPV
PPA > PALV < PPV

Ventilation-Perfusion Matching.

Causes of hypoxemia are hypoventilation, diffusion and shunting. Among these most common which occurs during anaesthesia is V/Q mismatch. Perfusion and ventilation are not uniform throughout the lung. As we move downwards from apex to base ventilation decreases and perfusion increases. This regional difference in gas exchange results in difference in gas exchange. The inspired air has a PO2 of 150mmHg and PCO2 is zero. The mixed venous blood entering the lung has PO2 of 40mmHg and PCO2 45mmHg. Thus the normal alveolar PO2 of 100mmHg and PCO2 of 40mmHG is determined by the supply and removal of these two gases at the alveolar level.

Respiratory exchange is not constant so we have a equation

V/Q = 8.63 R (Cao2-Cvo2)/PACo2 this is ventilation perfusion ratio equation.

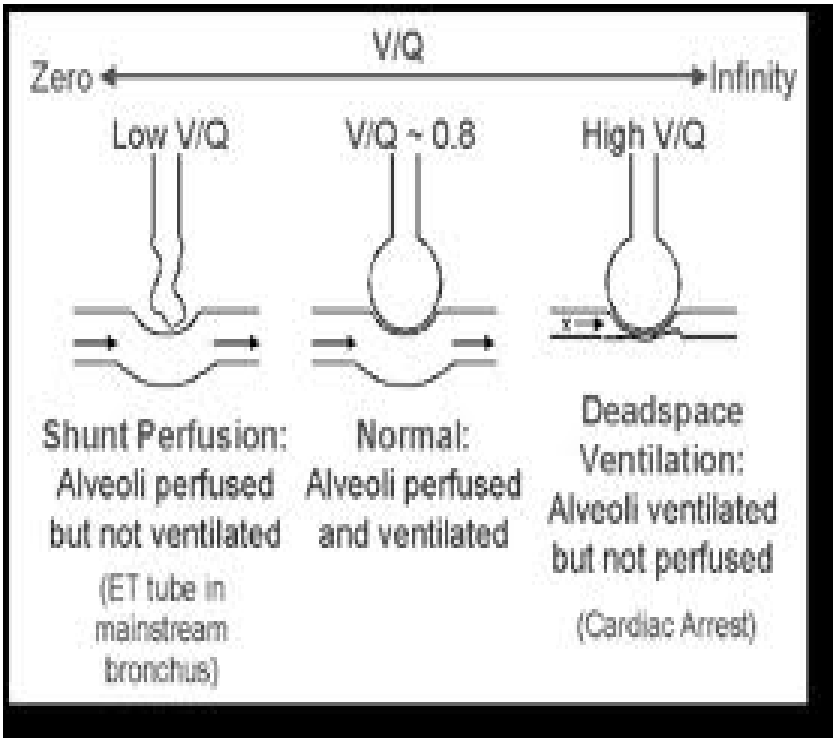
FROM THIS EQUATION

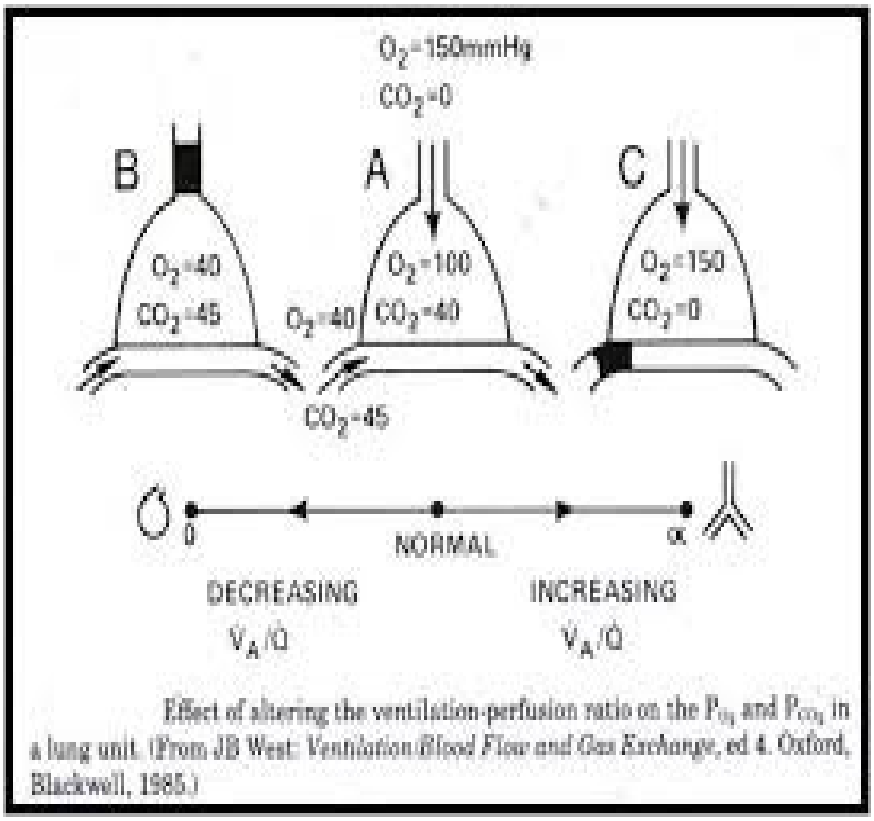
1. When ventilation becomes zero v/q becomes zero means O2 and CO2 concentration same as mixed venous blood
2. When v/q is gradually increased at one point perfusion is zero v/q becomes infinity means O2 concentration is high and that of CO2 is low reaching the inspired gas concentration.

Thus it is concluded when V/Q is altered its gas composition reaches that of either mixed venous blood or inspired gas.

High v/q mismatch means less PaO2 low v/q mismatch means more PaO2 Elimination of CO2 is affected by altered v/q ratio. It is overcome by increasing the alveolar ventilation but hypoxemia due to altered v/q ratio is not corrected by improving ventilation because these two gases follow a different dissociation curves.

V/Q inequality is measured by difference in alveolar arterial PO2.





Effect of altering the ventilation-perfusion ratio on the P_{O_2} and P_{CO_2} in a lung unit. (From JB West: *Ventilation/Blood Flow and Gas Exchange*, ed 4. Oxford, Blackwell, 1985.)

Perfusion across capillaries:

This is determined by pulmonary vascular resistance, alveolarO2 concentration and gravity. The response to low PO2 in alveoli is hypoxic pulmonary vasoconstriction.HPV possible mechanisms are

1. Release of vasoconstrictors in response to low PO2
2. Direct effect of low PO2 on pulmonary vessels.

The pulmonary arterioles have thick walls with rich smooth muscles.

The critical value of alveolar PO2 for HPV to occur is 70mmHg but above 100mmhg only little change is observed ..HPV is a protective mechanism

which directs the blood away from the poorly ventilated areas to well ventilated areas thus maintaining arterial o2 concentration.

Many anaesthetic drugs inhibit (vasodilators inhalational agents, nitric oxide high ,inspired O2 conc) and promote the HPV(hypoxia high altitude adrenergic agents vasoconstrictors).

In fetus only 15% cardiac output reaches the pulmonary vascular bed so less perfusion and low po2 in alveoli thus high vascular resistance is seen during fetal life .After birth inspired 02 co-centration increases and pulmonary vascular resistance falls dramatically so perfusion increases.

So HPV can be reversed by increasing the o2 supplementation.

Similarly low pH causes vasonstriction especially during coexisting hypoxia, but autonomic system has weak influence over the pulmonary vasculature .Pulmonary resistance is low and falls when CO increases because of capillary distension, resistance increases at high lung volumes and very low lung volumes it is believed that nitric oxide plays an important role in HPV mechanism.

Ventilatory Factors Affecting Alveolar Gas Composition

TRANSPORT OF O2 AND CO2:

TRANSPORT OF O2:

O2: it exists in two forms dissolved form and combined form with haemoglobin.

DISSOLVED FORM: It accounts for about 0.003ml for each 100ml of blood at 100mmhg of PAO2. This is not sufficient during exercise and in certain conditions due to increased O2 consumption and CO. thus increasing the inspired O2 concentration we can improve the dissolved O2 concentration thus the O2 demand states.

HAEMOGLOBIN : it contains four porphyrin rings and aminoacid chains. Here the iron is in ferric state. Ferrous state is seen in meth Hb. Difference in amino acid chains results in different types of Hb. HbA is adult form and HbF is fetal form the presence of gamma chain makes the Hb more affinity for O2.HbS has abnormal AA chain which sickles in the deoxygenated states.

O2 dissociation curve: the combination of O2 with Hb is reversible. Dissolved o2 concentration is 0.003ml in 100ml of blood. around 50mmhg of po2 o2 rapidly combines with Hb. after that the curve becomes flatter this gives a sigmoidal shape for the curve from the dissolved 02 we can calculate the combined oxy Hb amount. the maximum amount of o2 that can combine with a Hb is called oxygen capacity.one gram of Hb can combine with1.39ml of o2 so 15gm of Hb in 100ml of blood carries 20.8ml of o2.

O2 SATURATION = o2 combined with Hb /o2 capacity x 100

In arterial blood and in venous blood o2 saturation is 97.6% 75% respectively.Moreover,po2 is 100mmHg and 40mmHg respectively.

significance of o2Hb is more in anaemic patient.

Total O2 conc of blood is affected here which is given by

0.003.po2+(1.39.Hb. satu/100)

FACTORS AFFECTING O2 dissociation curve:

shift to left-increase in pH temp H+ ,decrease in CO2 and concentration of 2,3 DPG.

Shift to right- when changes occurs in opposite direction also anaemia hypoxia high altitude

BHORS EFFECT: effect of CO2 in unloading O2 to the tissues is called bhors effect.in tissues due to the metabolism releases CO2 ,acids which causes decrease in PH and release of O2.

CO dissociation curve: is same as O2 dissociation curve except that it has high affinity 240 times higher than O2 it means the partial pressure of pCO is much lower i.e. at pCO of 0.16mmHg 75% of Hb has combined with CO. Thus prevents O2 from combining with Hb. This affects the left shift of O2 dissociation curve in cyanide poisoning.

CO2 TRANSPORT:

Co2 is carried in three forms: dissolved ,bicarbonate and as caramino compounds.

DISSOLVED CO2:co2 is 20 times better soluble than O2 both CO2 and O2 follows the Henrys law.so this form of CO2 plays an important role CO2 transport.

BICARBONATE: CHLORIDE SHIFT

CO2 +H2O forms H2CO3. this reaction is more in RBC as red cells are rich in CA enzyme.

In the RBCs ,H2CO3 dissociates to form H+ + HCO3-.

HCo3 ion moves out of the red cell but H+ cannot move out as red cell is impermeable to H+ ion. So in order to maintain electrical neutrality Cl – ion moves inside the red cell. This follows GIBBMANNS DONNAN equilibrium.

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The liberated H+ ion combines with reduced Hb to form HbH+

Thus reduced Hb accepts co2 in peripheral tissues and oxygenation in lungs helps in liberating CO2 in lungs

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RESPIRATORY ACIDOSIS:

increase in co2 decreases the HCO3 /PCO2 ratio.Dissociation carbonic acid produces increase in HCO3- ion so if still persists kidneys starts conserving HCO3- ion. This is compensated respiratory acidosis. Renal compensation is determined by base deficit.

Increase in co2 is seen in hypoventilation and v/q mismatch.

RESPIRATORY ALKALOSIS: decrease in CO_2 concentration increases the HCO3 /PCO_2 ratio. Kidneys start excreting the bicarbonate ion if this persists to maintain the normal ratio. There will be a negative base excess/ base deficit.

Decrease in PCO_2 seen in hyperventilation, high altitude

METABOLIC ACIDOSIS: decrease in HCO_3- ion decreases the HCO_3 / PCO_2 ratio. Respiratory compensation occurs by decreasing the PCO_2 by hyperventilation to maintain the normal ratio. Here also there is a base deficit. Hyperventilation is due to increase in H+ ion on chemoreceptors.

This is seen in DKA, lactic acidosis, tissue hypoxia

ADVERSE EFFECTS OF METABOLIC ACIDOSIS

Metabolic acidosis produces a number of complications in various organs such as cardio vascular system, central nerves system and renal system. In metabolic acidosis there is initially increase in the stroke volume and cardio contractility due to the effect of catecholamines in a PH of 7.4 - 7.2 and maintenance of normal blood pressure. When the PH goes below 7.2 there is decreased sensitivity of catecholamines to the heart resulting in poor ionotropic and contractility function and there is profound vasodilatation resulting in hypotension. As metabolic acidosis progresses there is myocardial in depression resulting in poor contractility and failing heart.

There is mental confusion and lethargy associated with decreased PH though studies show no change in CSF biochemistry. There is PH dependant decrease in oxygen affinity and decreased 2, 3 DPG causing a change in oxy haemoglobin dissociation curve.

In chronic metabolic acidosis as seen mostly with CKD there is increased potassium concentration which pre disposes to dysrrythmias. There is decreased lynfosites production and decreased immune response and increased inflammatory reaction seen with metabolic acidosis. Studies have found that there is PH dependant decrease in insulin response in acute cases.

In the muscular skeletal system there is increased bone resorption reading to growth retardation in children. Sympathetic over activity is also seen with many cases of metabolic acidosis.

The respiratory compensation is seen in the form of hyper ventilation. There is also decreased pre-load and after load to the heart leading to hypotension.

METABOLIC ALKALOSIS:

Increase in HCO_3 increases the HCO_3/PCO_2 ratio. Respiratory compensation occurs by increasing the PCO_2 by hypoventilation or decrease in alveolar ventilation. There will be increase in base excess. This is seen in vomiting, diarrhea, alkali ingestion.

The co2 elimination is easier compared to promote the oxygenation because co2 diffusion is 20 times higher. The tissues use oxygen to a critical point of PO2 at mitochondria around 3mmHg after that anaerobic metabolism supervenes and results in lactic acidosis. Also in some tissues the PO2 is 5mmhg so the purpose of providing continues inspired oxygen (and continuous blood flow to tissues is also necessary) is to keep the PaO2 at high level .Thus maintaining a concentration gradient so that O2 diffuses into the tissue continuously thus maintaining the aerobic metabolism.

Composition of Alveolar Gases

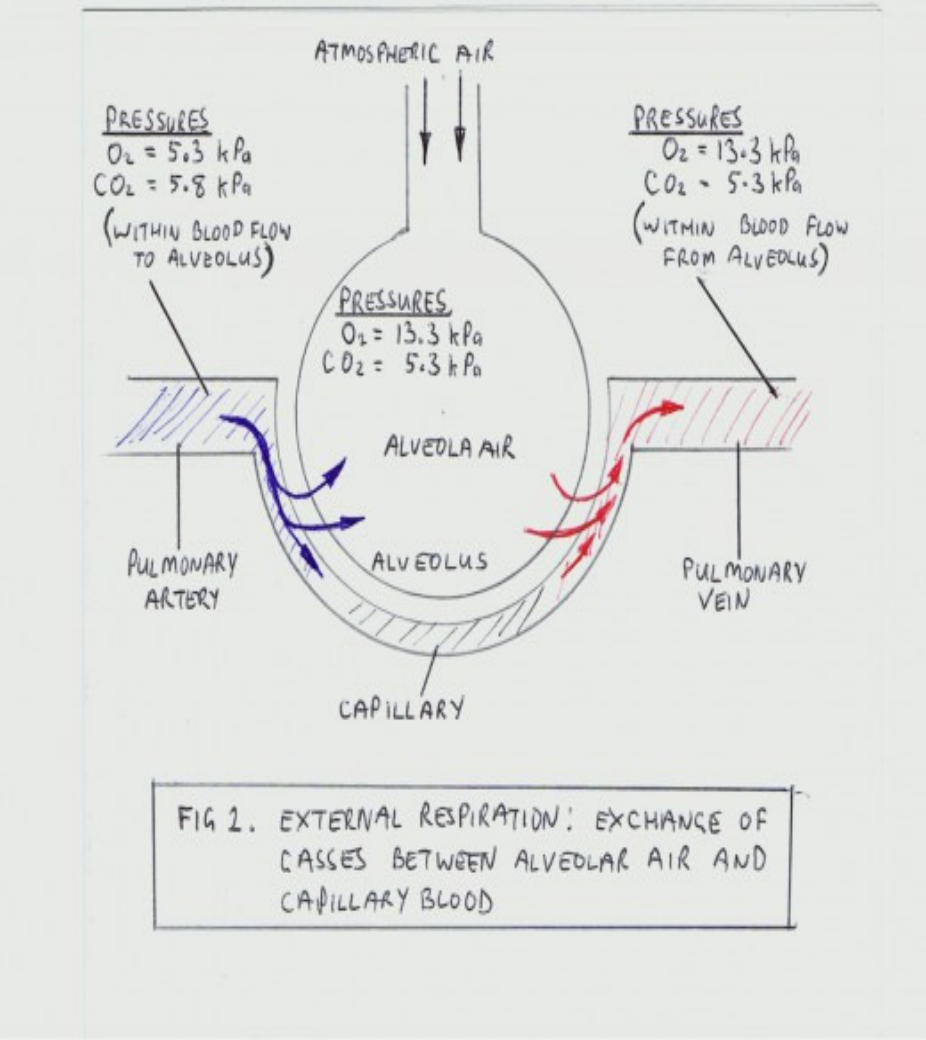


FIG 2. EXTERNAL RESPIRATION: EXCHANGE OF GASSES BETWEEN ALVEOLAR AIR AND CAPILLARY BLOOD

Effect of ventilation perfusion ratio on alveolar gas concentration:

V/Q ratio is low in the base.it means high perfusion at the base but less ventilated that is low inspired oxygen concentration at the alveolar level but high PaCo2 at capillary level in that region.

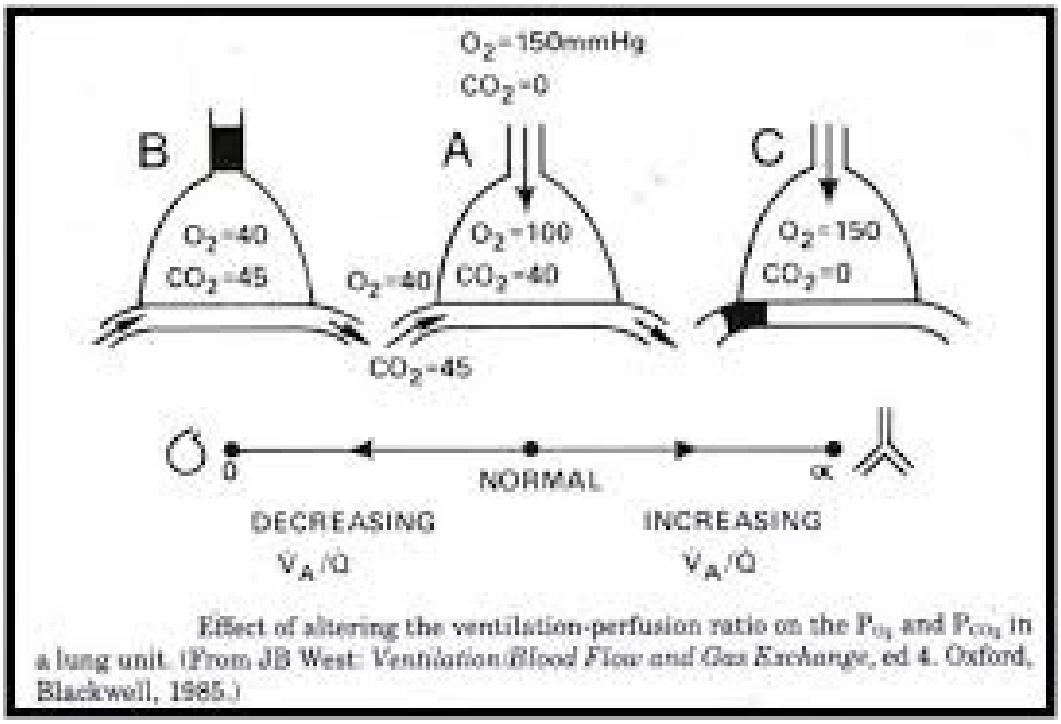
V/Q ratio is high in the apex.it means high ventilation at the apex but less perfused that is high inspired gas concentration at the alveolar level but low PaCo2 at the capillary level in that region.

In other words low V/Q ratio, $_{PAO2\ i}$s less and $PaCO_2$ is more

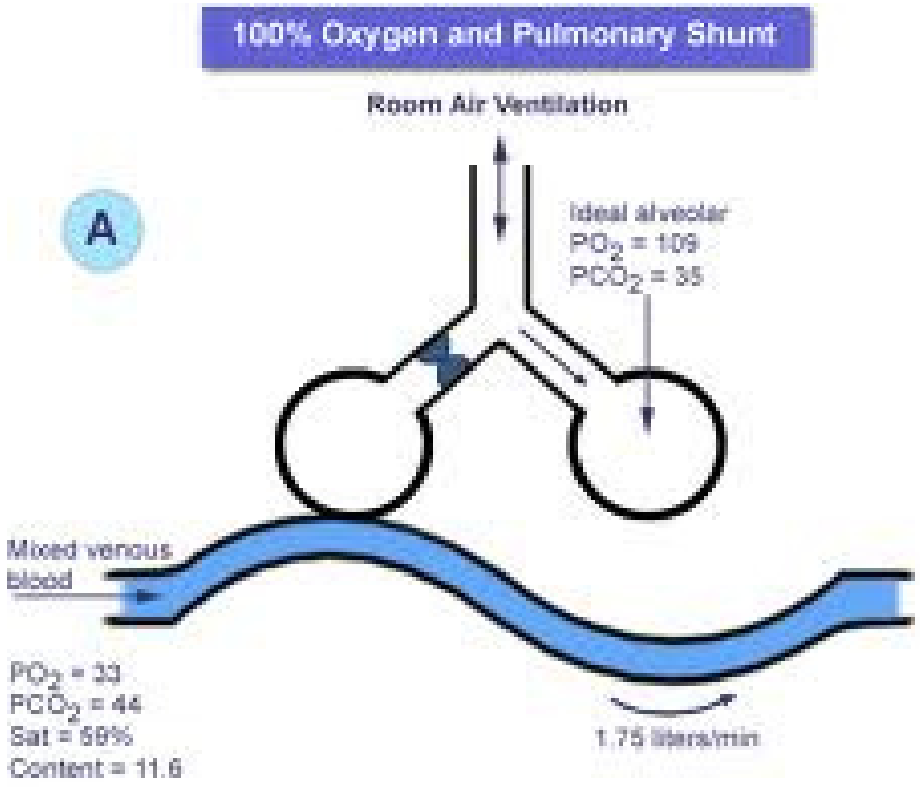
High V/Q ratio, PAO_2 is more and $PaCO_2$ is less

This is mainly seen in the upright position. Hence changes in position alter the v/q ratio which in turn affects the alveolar gas concentration. So increase in arterial side pco2 can be removed by increasing the inspired oxygen concentration at the alveolar level at low v/q ratio state. But in high v/q ratio this is not applicable because inspired oxygen concentration is already high only perfusion is defect so need to improve the perfusion at this region.

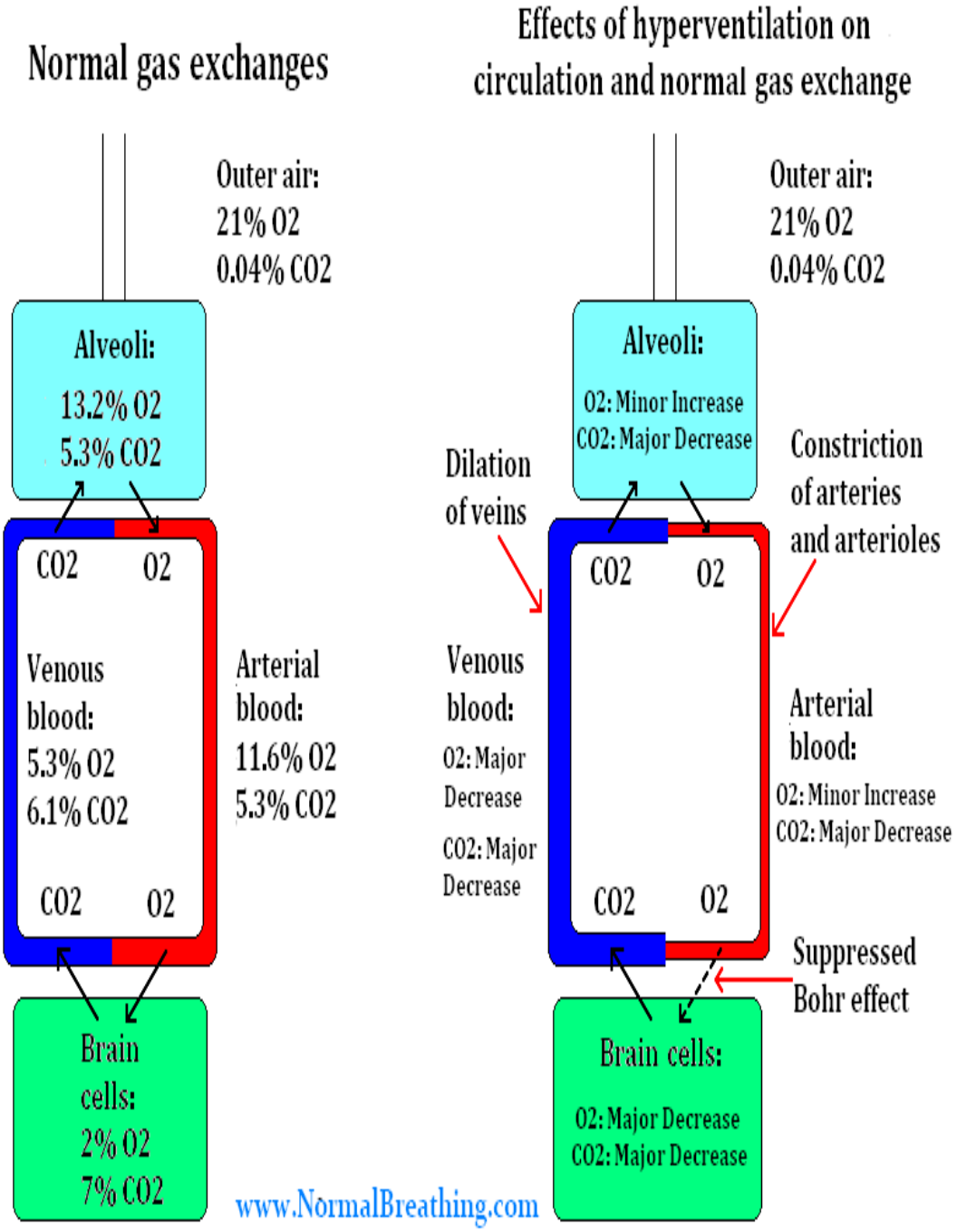
In other words in shunting due to hypoxia can be eliminated by giving the 100% O2 we are increasing the PAO2 in the inspired air thus preventing HPV in that region better elimination of CO2 and improves oxygenation of blood. This is more useful in high PaCO2 production states.



Effect of altering the ventilation-perfusion ratio on the P_{O_2} and P_{CO_2} in a lung unit. (From JB West: *Ventilation/Blood Flow and Gas Exchange*, ed 4. Oxford, Blackwell, 1985.)



Transport of O2 and CO2



Transport of O2 in blood:

O2:it exists in two forms dissolved form and combined form with haemoglobin.

DISSOLVED FORM: It accounts for about 0.003ml for each 100ml of blood at 100mmhg of PAO2 .This is not sufficient during exercise and in certain conditions due to increased O2 consumption and CO. thus increasing the inspired o2 concentration we can improve the dissolved o2 concentration thus the O2 demand states.

HAEMOGLOBIN: it contains four porphyrin rings and aminoacid chains. Here the iron is in ferric state. Ferrous state is seen in meth Hb. Difference in aminoacid chains results in different types of Hb. HbA is adult form and HbF is fetal form the presence of gamma chain

makes the Hb more affinity for O2.HbS has abnormal AA chain which sickles in the deoxygenated states.

O2 dissociation curve : the combination of O2 with Hb is reversible. Dissolved o2 concentration is 0.003ml in 100ml of blood.around 50mmhg of po2 o2 rapidly combines with Hb.after that the curve becomes flatter this gives a sigmoidal shape for the curve from the dissolved 02 we can calculate the combined oxyHb amount.the maximum amount of o2 that can combine with a Hb is called oxygen capacity.one gram of Hb can combine with1.39ml of o2 so 15gm of Hb in 100ml of blood carries 20.8ml of o2.

O2 SATURATION = o2 combined with Hb /o2 capacity x 10

In the arterial blood and in the venous blood o2 saturation is 97.6% and 75% respectively and po2 is 100mmHg and 40mmHg respectively.

significance of o2Hb is more in anaemic patient.Total O2 conc of blood is affected here which is given by 0.003.po2+(1.39.Hb. satu/100).

Transport of CO2 in blood.

is carried in three forms:dissolved ,bicarbonate and as caramino compounds.

DISSOLVED CO2:co2 is 20 times better soluble than O2 both co2 and o2 follows the Henrys law.so this form of CO2 plays an important role CO2 transport.

BICARBONATE:**CHLORIDE SHIFT**

CO2 +H2O forms H2CO3 this reaction is more in RBC as red cells are rich in CA enzyme. In the RBCs ,H2CO3 dissociates to form H+ + HCO3- .

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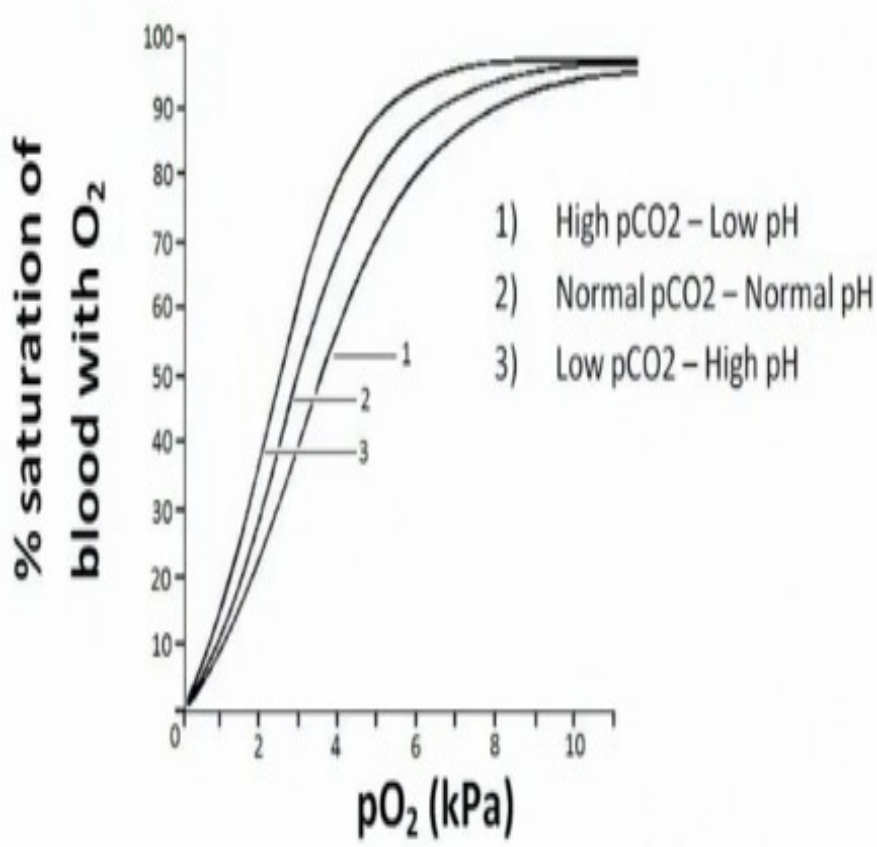
Shape of the Oxy-Hb Dissociation Curve.

It is a sigmoid shaped curve representing the release of oxygen from Hb by various factors.



Shifts in the Oxyhemoglobin Dissociation Curve

FACTORS AFFECTING O2 dissociation curve:

Shift to left-increase in pH temp H+, decrease in CO2 and concentration of 2,3 DPG. Shift to right when changes occurs in opposite direction also anaemia hypoxia high altitude.

BHORS EFFECT: effect of co2 in unloading o2 to the tissues is called bhors effect.in tissues due to the metabolism releases co2, acids which causes decrease in ph and release of o2.

CO dissociation curve: is same as o2 dissociation curve except that it has high affinity 240 times higher than o2 it means the partial pressure of pCO is much lower i.e. at pCO of 0.16mmHg 75% of Hb has combined with CO. thus prevents O2 from combining with Hb.

This affects the left shift of O2 dissociation curve in cyanide poisoning.

Hypoxia:

It is decrease in the partial pressure of O2 in the atmosphere at any level between the supply and demand the body. It is said to occur when PO2 is less than 100mmHg in the alveolar

Less than 60mmHg in the blood level

Less than 5mmHg in the tissue level

Hypoxemia is decrease in the O2 content in the arterial blood. Normally it occurs when the partial pressure O2 in blood is less than 60mmHg.Hypoxia can occur without hypoxemia also.

TYPES OF HYPOXIA: four types

1. HYPOXIC HYPOXIA:

Due to deficiency of oxygen being absorbed into the lung.

Thus it may be due to decrease in the partial pressure of O2 in the inspired gas (/its complete absence called anoxic hypoxia) or due to some pathology in the alveolar capillary membrane which prevents the diffusion of O_2 into the alveolar capillaries.

Eg: high altitude (physiological hypoxia) Smoking(also increases the carboxy Hb level in the blood)

Pneumonia ,

interstial lung disease,

asthma,

pulmonary edema

In anaesthesia, diffusion hypoxia is a type of hypoxic hypoxia due to nitrous oxide. This is prevented by increasing the partial pressure of O2 in the inspired gas i.e by giving 100%oxygen.

ANAEMIC HYPOXIA:

Due to decrease in O2 binding capacity of Hb mainly due to decrease in the amount or quality of Hb. Hb is the main carrier of O2 to the tissues. Thus in anaemia affects the total O2 content of blood, which is more important during the period of high demand in the tissues for O2 like exercise.

Eg: CO poisoning

sickle cell anemia

Acute or chronic blood loss

Aspirin, sulphonamides, nitrates

Methhaemoglobinemia

In normal patients Hb % needed for anaesthesia is 8gm% In cardiac patient and critically ill patient this number increases to 10gm%

This is prevented by improving the Hb content either by improving the iron and vit B12 content or in emergency by blood transfusion and treating the cause of anaemia.

2. **STAGNANT HYPOXIA:**

Due to decrease/cessation of blood flow to the tissues indirectly affects the O2 supply. Eg: cardiac failure,

cardiac arrest.

Decreased circulatory blood volume CPPV

This is prevented by treating the underlying conditions and by ionotropes to improve the peripheral circulation hence the o2 supply to the tissues.

3. HISTOTOXIC HYPOXIA:

due to the defect in O2 utilization or extraction by the tissues

Eg: cyanide poisoning

Narcotics

Alcohol

Prevented by treating the cause.

PHYSIOLOGY OF TOURNIQUET APPLICATION

Application of a high pressure air filled cuff to a limb is used to prevent the central circulatory spread of local anaesthetic agent during intravenous regional anaesthesia. It is also used to reduce the bleeding in the surgical area and improved vision of the surgical field. But using such a high pressure tourniquet can cause profound physiological changes in the body which mainly depends on the duration of tourniquet application and the preoperative general condition of the patient.

Tourniquet application

To maintain a clean bloodless field during surgery

The width of the cuff should be more than half of the width of the particular limb .The edges of the cuff should overlap so that it distributes its pressure uniformly in all the Ares of the limb circumference. Ideally the overlapping area of the tourniquet should be away from the main neurovascular bundle of the limb concerned. After the application of tourniquet the limb should be elevated above for one minute or by the application of Esmarch bandaging or a Rhys-Davies exsanguinator. (In infection or tumours of the limb, exsanguination by methods other than limb elevation is contraindicated.)

When tourniquet is applied on the lower limb the inflation pressure is about 100mmhg more than the systolic BP while in the upper limb it is 50 mmhg more than the systolic BP. The pressure required for compression depends mainly on the bulk of the underlying muscle to be compressed. While

cleaning the limb the cleaning solution should not come in contact with padding of the tourniquet. The iodine and alcohol present in the cleaning solution can cause skin irritation and the diathermy used during surgery can cause burns if the padding is soaked with the cleaning solution.

Physiological changes

Cardiovascular

Application of tourniquet increases the distal pressure in the limb and leads to increase in systemic blood pressure, central compartment venous pressure, increase in heart rate. All these effects are due to increase in the systemic blood volume of about 15% due to compression of the capacitance vessels. Opposite hemodynamic changes occur during deflation of the tourniquet due to reduction in the systemic vascular resistance and resumption of venous blood flow. Regional anaesthesia to some extent can reduce this adverse outcome. Etamine at a dose of 0.25mg/kg can significantly reduce this dynamic changes.

Patients who are cardiovascularly stable may exibit a rise in systemic vascular resistance after 1 hour of inflation. but it may continue to rise even sfter effective measures for several hours. They will respond as fall in blood pressure only after tourniquet removal.

Severe hypertension and impending limb ischemia are the major limiting factors for tourniquet deflation. The exact mechanism for this delayed rise in blood pressure is still unclear. There is increase in the blood flow to the

particular limb after deflation for about 15 mins. This is due to the release of anaerobic metabolites that cause reactive vasodilatation in capillaries in the muscle.

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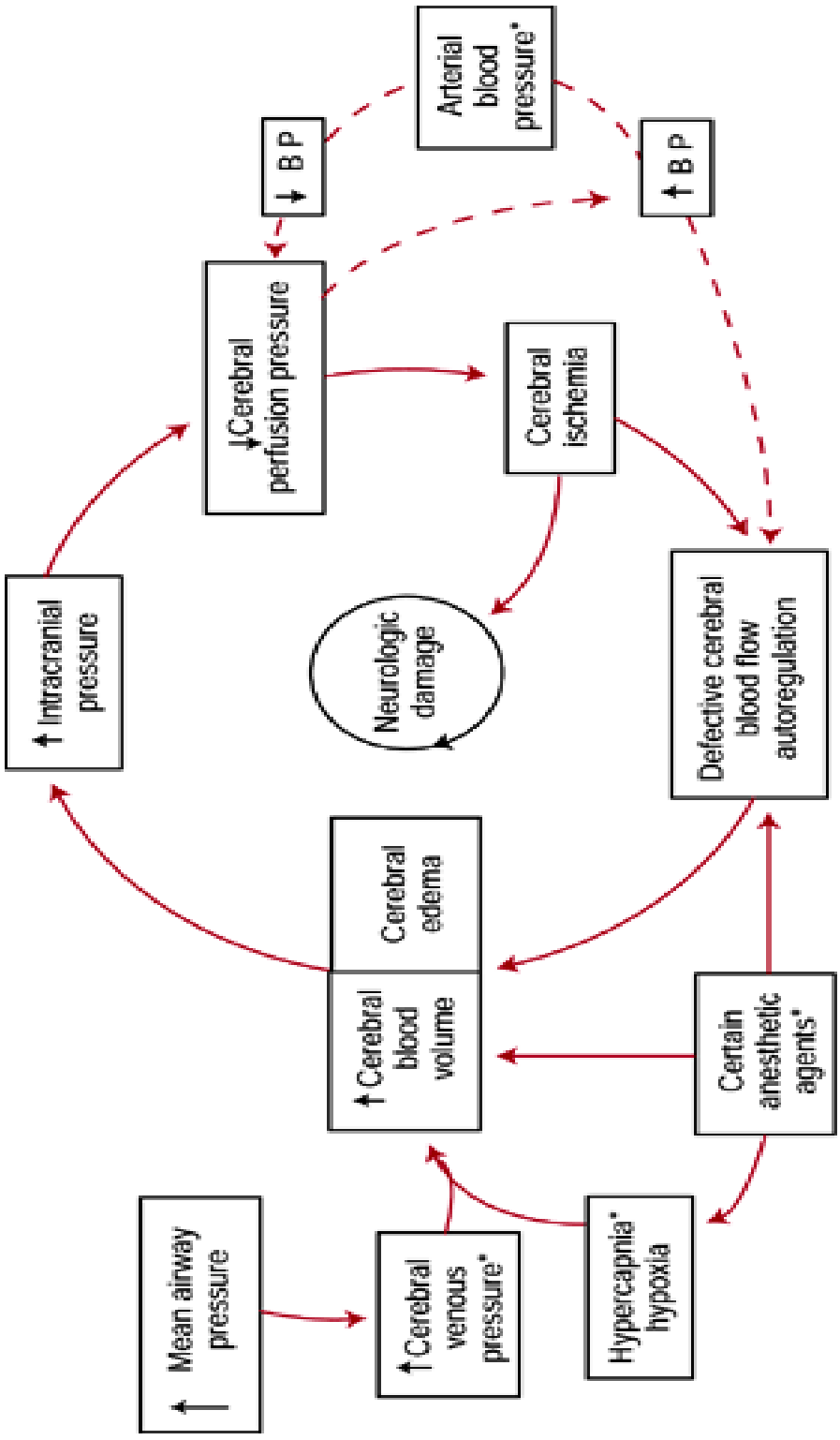
Cerebral circulatory changes

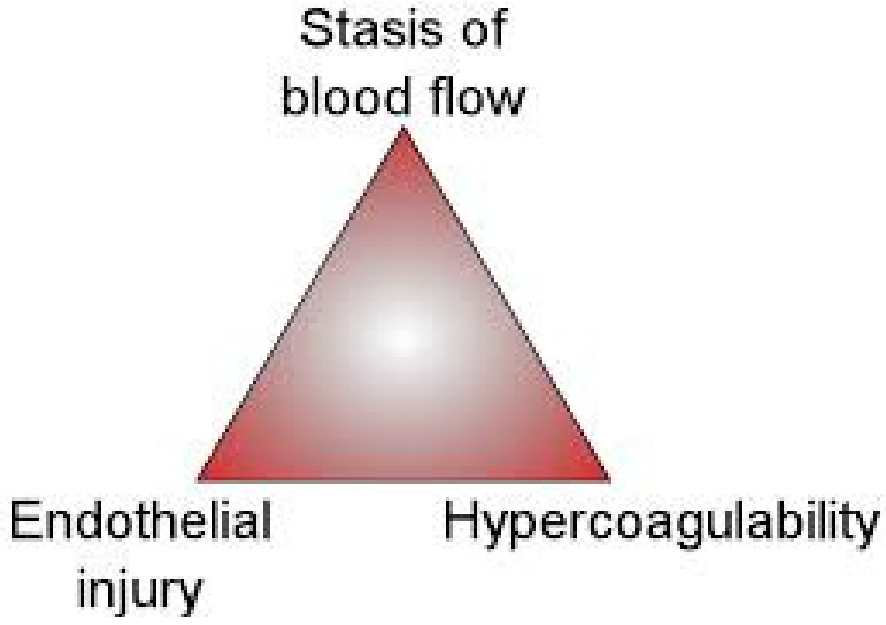
The increase in Fraction of carbondioxide in the blood reaching the cerebral circulation causes increase in the middle cerebral artery blood flow increasing the intracranial pressure during the release of tourniquet. This increase in the cerebral blood volume causes damage to the brain in patients who already has increased intracranial pressure.

Polytrauma patients with head injury should be monitored cautiously when their associated limb injuries are treated with tourniquet. Hyperventilating the patient thereby decreasing the carbondioxde drive can decrease the cerebral blood flow and thereby the intracranial pressure to some extent.


Arterial blood pressure*
↓ B P
↑ B P
↓Cerebral perfusion pressure
Cerebral ischemia
↑ Intracranial pressure
Neurologic damage
Defective cerebral blood flow autoregulation
Cerebral edema
↑Cerebral blood volume
Certain anesthetic agents*
↑ Mean airway pressure
↑Cerebral venous pressure*
Hypercapnia* hypoxia

Haematological effects

Applying a tourniquet can cause hypercoagulability and fibrinolysis though surgery itself can cause both irrespective of tourniquet. Both pain of surgery and of tourniquet can increase the release of catecholamines which then causes hypercoagulability of blood. The application of an Esmarch bandage also causes tissue compression thereby increased platelet aggregation .After tourniquet inflation the resultant ischemia causes the release of tissue plasminogen activator, which activating the antithrombin III and thrombomodulin–protein C anticoagulant causing systemic thrombolysis when the tourniquet is released . It is also seen that acidosis and anoxia due to tourniquet cause tissue plasminogen activator release although there is no relation between the duration of ischaemia and the degree of fibrinolysis has been obtained .



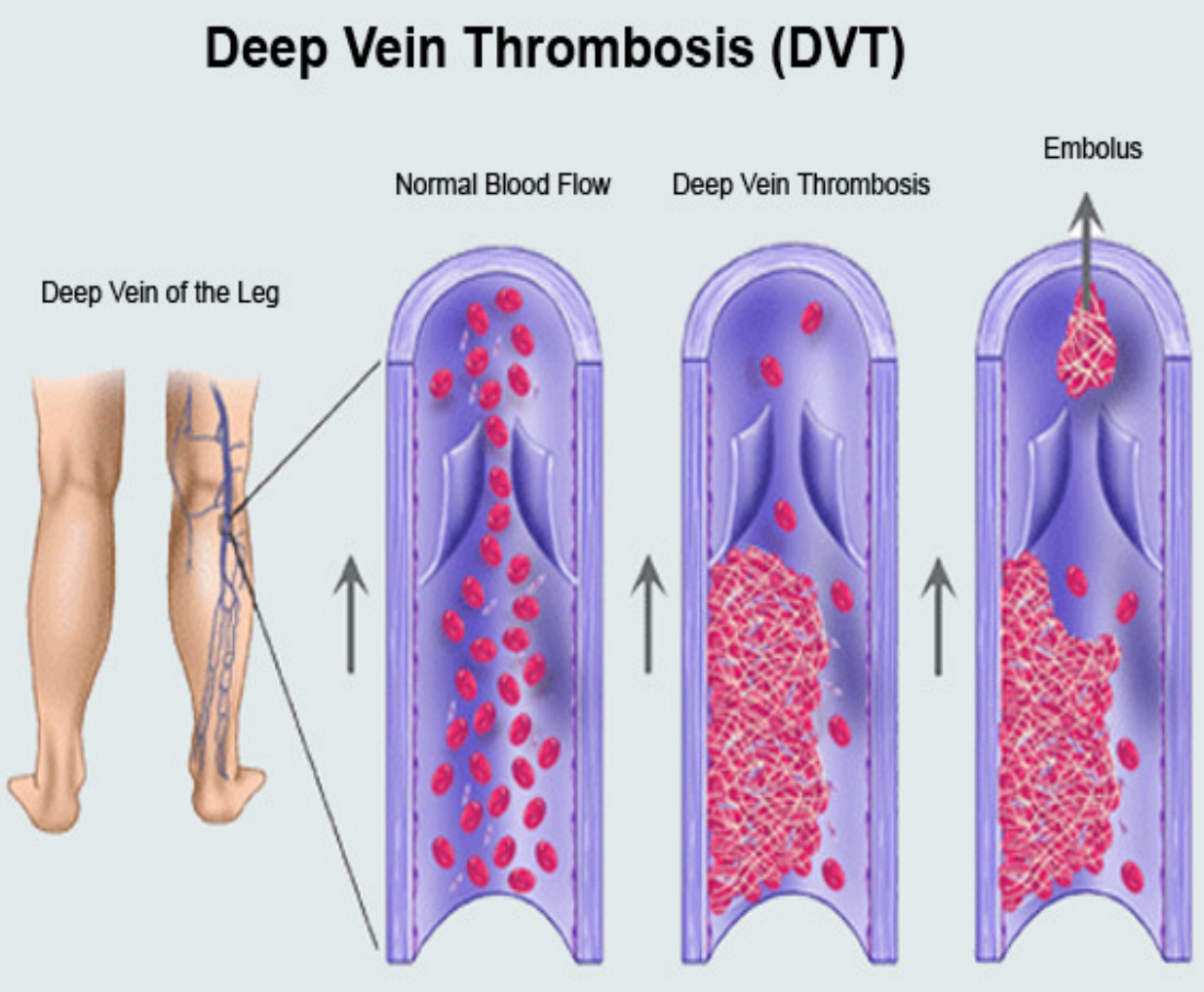
Applying a tourniquet can cause hypercoagulability and fibrinolysis though surgery itself can cause both irrespective of tourniquet. Both pain of surgery and of tourniquet can increase the release of catecholamines which then causes hypercoagulability of blood. The short period of hypercoagulability as explained above is responsible for the incidence of post tourniquet bleeding . Though the incidence of deep vein thrombosis after tourniquet application is 17-54% it is seen that tourniquet itself does not cause increased deep vein thrombosis.

A large number of trials revealed that using tourniquet and its inflation and deflation has caused a number of incidents of fatal pulmonary embolism. So some people suggest that tourniquet can be labelled as contraindicated in high risk group of patients. These high risk group of patients are patients who are on prolonged immobilisation, patients who has a history of deep vein thrombosis, morbidly obese patients.

Sevaral miliary microemboli are seen in the chest x rays of patients in tourniquet after deflation which suggests they are due to reperfusion from the ischemic limb. Fatal pulmonary embolism occurs after intramedullary guide wire insertion, femoral reaming and cementing of long-stemmed femoral prostheses, showing that surgical instrumentation of the medullary cavity releases these micro emboli. In a study it was found that in patients who underwent Total knee replacement under general anaesthesia, many of them showed large and smaller emboli on transesophageal echo indicating their origin

from the medllary nailin. A study on the inci of venous emboli during total knee arthroplasty without tourniquet detected small and large venous emboli in 50% and 30% of patients, respectively.

Multiple logistic regression analysis shows that there is 5.33 fold increase in the incidence of pulmonary embolism in tourniquet used total knee arthroplasty . Low-dose LMWH has a role in preventing the emboli.1000units IV before tourniquet application,1000 units given during skin incision,500 units given during preparation of femoral canal in total knee replacement can prevent large emboli being generated.


Deep Vein Thrombosis (DVT)
Embolus
Normal Blood Flow
Deep Vein Thrombosis
Deep Vein of the Leg

Temperature

Inflation of the tourniquet makes the limb cold and puts it to hypothermia. It has some advantages of reduced metabolic rate as well. During deflation of the tourniquet the cold blood enters the circulation reducing the core temperature to 0.6 degrees for every hour of tourniquet inflation.

Metabolic changes

Application of tourniquet for more than 30mins causes the release of free radicals, hypoxemia, hypercarbia, mixed acidosis. These changes are however well tolerated by healthy individuals. But it may be detrimental to patients with poor cardiopulmonary reserve.

Hyper ventilation to some extent can compensate for this hypercarbia after tourniquet deflation. When taken under general anaesthesia rather than using inhalational agent propofol infusion can be used for maintenance of anaesthesia. Bilateral tourniquet application should be avoided. If warranted atleast simultaneous inflation of the tourniquet is avoided.

Complications

The appearance of complications are directly related to the inflation pressure and the duration of inflation of the tourniquet.

Limb ischaemia

It is the most serious complication. It is directly propotional to the tourniquet duration. The average duration lies between 30 mins to 4 hours.Studies recommend that duration for more than 30 mins shows the onset of anaerobic metabolism. After 1 hour application of tourniquet, electron microscopy pictures show evidence of depleted glycogen stores in the sarcoplasm of the musle fibres.2 hours later to tourniquet application evidences of acidosis such as mitochondrial swelling, disappearance of z-lines in the muscle spindles are seen. The safety limit of tourniquet application can be increased by reperfusion and exsanguination in order to reduce the anaerobic metabolism. This has the disadvantage of supplying more substrate for the anaerobic metabolism.

Pressure-related nerve damage

Excessive pressure applied by the tourniquet can cause rupture of the Schwann cell membrane in the nerves which causes neuralgia parasthetica. But it resolves within few weeks or months.

Excessive compression of the tourniquet can compress the neurovascular bundle leading to the formation of micro emboli in the exsanguinated limb. The formation of micro emboli can increase the incidence of pulmonary

microembolic occlusion. Some may even go for mechanical ventilation post operatively due to respiratory failure especially in trauma patients. In a healthy patient with proper exsanguination, no clinical evidence of acidosis and emboli occurs usually. The incidence of deep vein thrombosis is high in surgeries where tourniquet is used. Thrombo prophylaxis may be given in all patients above 40 years undergoing tourniquet used surgeries except in knee arthroscopic surgeries. It can also be given in patients who are at high risk for DVT.

Tourniquet pain

It occurs about 45-60mins after tourniquet application. It can present as severe unbearable pain. Initially it starts as dull aching pain after about 30 mins of tourniquet inflation. The patient may feel excruciating pain in the exsanguinated limb though the anaesthesia may be satisfactory otherwise. This tourniquet pain is transmitted through unmyelinated C type fibres which are responsible for the transmission of dull aching poorly localised pain and they are resistant to local anaesthetics. The myelinated A delta fibres are better blocked by local anaesthetics and they carry sharp pain. Initially when the bloc is given, the local anaesthetic block both the fibres . Later when the local anaesthetic is metabolised, C type fibres show pain while the A delta fibres are still blocked.

It is difficult to treat the tourniquet pain once it has developed. It can be relieved only on releasing the tourniquet. Sometimes General anaesthesia may

be warranted for this pain. Opiods supplementation are disappointing as their toxicity reveals once the procedure is over and the afferents of tourniquet pain are terminated inspite of them controlling the pain poorly.

Hypertension:

Patients during surgery under tourniquet show hypertension which are not controlled by opiods. The reason for this hypertension is unclear but it can be attributed to acidosis, hypercabnia, hypoxia etc. However the episode is terminated after the release of tourniquet.

Muscle injury

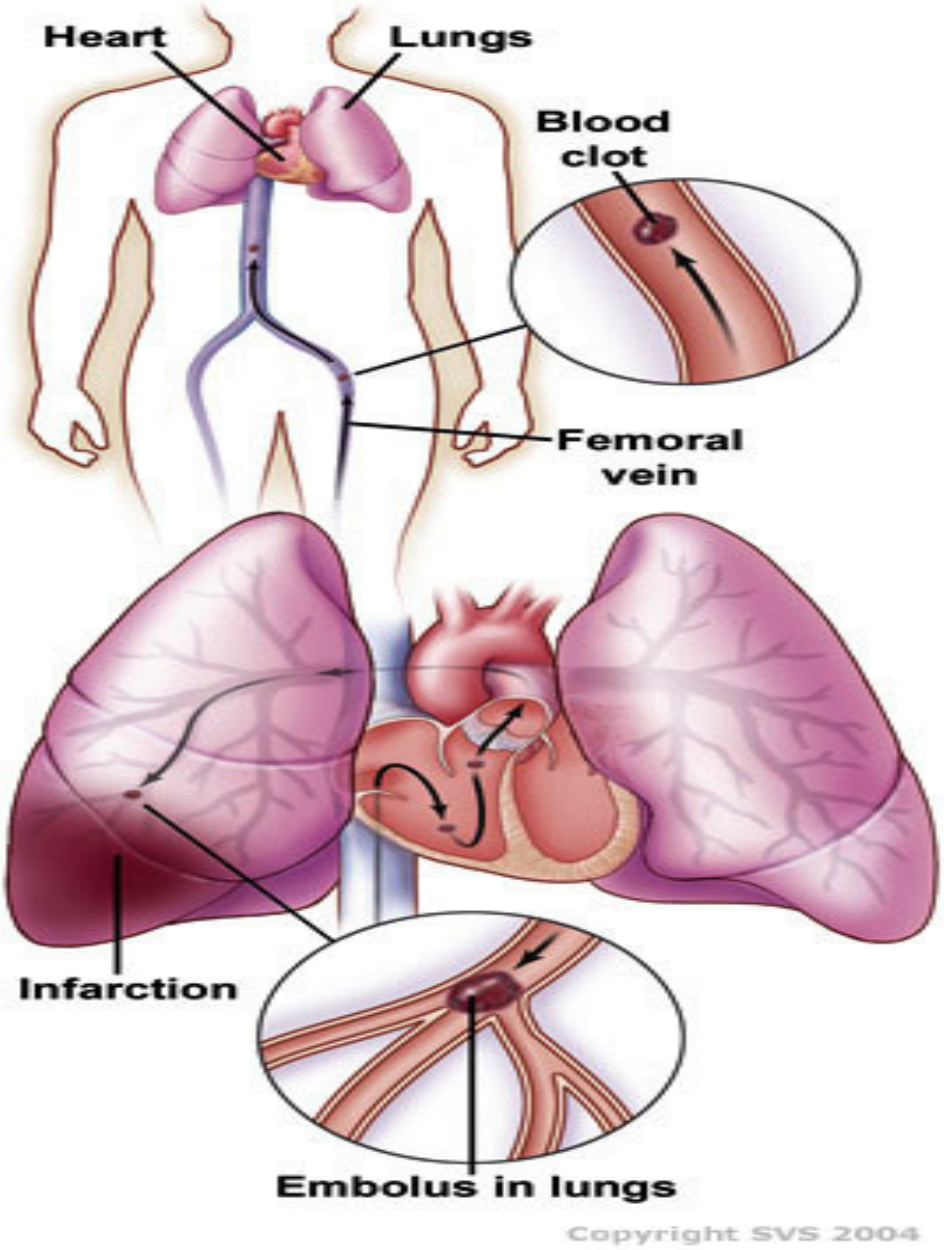
Tourniquet application causes muscle injury immediately beneath it and also distal to its application. This is due to muscle compression, vascular compression, nerve damage and absent of blood supply distal to the tourniquet and its resultant ischemia. In both animal and human studies, it was found out that there is decreased oxygen tension, ATP, creatine phosphate and glycogen in the area beneath the tourniquet and there is acidosis, increased carbodioxide tension, lactic acid, in that area.

The time taken for the cell to recover from the metabolic insult depends on the duration of tourniquet application. It indirectly correlates with the time for the ATP replenishment to occur that would have been lost during the episode of limb ischemia. During the ischemic period toxic free radicals are generated in the form of hydrogen peroxides in the ischemic tissues.

All the features such as numbness, ischemia, microvascular congestion due to tourniquet leads to post tourniquet syndrome. Symptoms of this syndrome include numbness of the limb, subjective parasthesia due to neuropraxia, subjective weakness of the limbs, pain etc. It was also found that the force produced by the muscles after tourniquet is much reduced and it will recover about 3 weeks post tourniquet time .It is also propotional to the tourniquet pressure applied. An advantage of fastened recovery is postulated in lower limb tourniquet surgeries

Vascular injury

Vascular injuries due to tourniquet are uncommon .If at all it happens it occurs most commonly in the lower limb surgeries. Rush et al.in his study has found out that inflation of the tourniquet crushes the atheromatous plaques in the atherosclerotic vessels. These fractured plaques also block the blood flow to the already atherosclerotic vessels. Kumar *et al.* , has done a study and found out that tourniquet usage can be contraindicated in patients who have absent distal pulses, atheromatous and calcified femoropopliteal vessels, and a past history of vascular limb surgeries .


Heart
Lungs
Blood clot
Femoral vein
Infarction
Embolus in lungs
Copyright SVS 2004

Skin injury

Skin injury is very uncommon in tourniquets. It usually occurs due to poorly applied tourniquet. It is commonly seen in children and elderly who has very fragile skin. It is basically due to spirit used for prewash seeping accidentally beneath the tourniquet causing frictional burns in the skin.

Tourniquet time and pressure

There is no concensus regarding the safe time and pressure of tourniquet application. In general nerves are more susceptible to tourniquet pressure and muscles are more susceptible to duration of tourniquet.

Studies have shown that an average of 1.5 to 2 hours is the optimal duration. Studies suggest that anaerobic metabolism sets in at duration of 30mins after tourniquet application. Newman in his study has found out that most of the muscle ATP, and glycogen stores are depleted when the tourniquet duration exceeds 1.5 to 2 hours. The recovery period after deflation mainly depends on the replenishment of the ATP stores after removal of tourniquet.

Wiggles studied the duration and its impact. He demonstrated a time dependent increase in acidosis in the venous blood of the limb under tourniquet. He also emphasised that duration of more than 1.5 to 2.0hours showed ultrastructural changes in the muscle fibres.

Hepenstall et al, and his associates demonstrated a similar results in dogs. Pedowitz *et al*.,in a similar way used technitium99 pyrophosphate as a indicator to prove similar results. He was also able to demonstrate Muscle fibre necrosis

in his study. Gersoff *et al*. studied it cats that tourniquet duration of even 1 hour produced subtle changes in the functional ability if the muscles that it persists for even about 7 days.

Some other methods have also been tried for prolongation of tourniquet time. It has been suggested that cooling the limb before tourniquet application can increase the tourniquet application time for up to 4 hours. However these studies have been done in animal models and human studies are yet to gain acceptance.

Neimkin & Smith studied the two cuff technique that involves inflating each cuff alternatively at hourly interval without a period of deflation of simultaneous cuffs. It helps in avoiding compression injury under a single cuff due to prolonged duration.

Nerve palsy due to tourniquet is the most dreaded complication of tourniquet. Direct pressure under the tourniquet and the shearing pressure at the edges of the tourniquet are the major culprits for this complications and must be seriously viewed.

Drug kinetics

Tourniquet application has its implications in drug kinetics also. Drugs that are administered before inflation are blocked within the sequestered limb and they are redistributed after tourniquet deflation. Similarly drugs that are given after tourniquet inflation are not distributed to the tourniquet limb and hence they have lower volume of distribution.

Schmitt *et al*.studied the effects of Fentanyl and midazolam on tourniquet application. Midazolam was administered before tourniquet inflation intravenously. It was seen that there was a sustained increase in serum midazolam after tourniquet release which is more pronounced in patients more than 70 years of age. It is significant in that the effect of the drugs can prolong into the post operative period exerting its effect seriously .

Similarly in a study vecuronium was administered before inflation and its effects persisted due to reperfusion after tourniquet deflation.

Tourniquet inflation also has its effect on antibiotic administration. Administered before application has poor penetration. An optimal time of 5mins is necessary for antibiotics before tourniquet inflation.

Tunstall studied the isolated forearm technique in patients undergoing caesarean section under general anaesthesia. One arm was isolated using tourniquet before administering drugs. Intraoperatively patients were given commands to use their tourniqueted limb. About 97% showed a positive result of obeying commands. But none of them exhibited recall. It is of little significance to monitor the depth of anaesthesia.

Tourniquet safety.

Most of the tourniquet related complications occur as a result of improper tourniquet use and poor technique. So they can be avoided by adequate practising .They can also occur as a result of machine failure. Both over inflation and under inflation cause problems. Over pressurised tourniquet

cause tissue necrosis while under pressurised tourniquets fail to achieve hemostasis and tend to facilitate the spread of local anaesthetic into the circulation in intravenous regional anaesthesia. Improper tourniquet application and improper practice can lead to skin blisters, chemical burns due to spirit, skin necrosis, cuff bruises etc.

Special attention should be given to check the quality of the tourniquet. While using a tourniquet we should check for its leak proof, changes in pressure readings over time, its pressure gauges should be calibrated properly, the rubber tubing's should be leak free. The surface of the tourniquet should also be clean ensuring frictional injuries free, hard under surfaced tourniquet should be avoided.

The tourniquets when applied should be monitore carefully. Frequet checking of the pressure leaks, tubing's should be done. The surgeons should be informed about the tourniquet time periodically in order to avoid prolonged ischemic time which posts the patient to limb ischemia.

Patients who are at risk of tourniquet related complications should be monitored carefully. Morbid obese patients are at risk of nerve injuries due to tourniquet owing to the smaller size of the proximal arm circumference when compared to the cuff.

PHYSIOLOGY OF BUFFERING MECHANISM.

There are several buffering mechanisms in the body that tightly regulate the physiological pH despite the daily net acid gain. Buffering is accomplished by chemical buffers, the lungs, and the kidneys.

Chemical buffering.

They are the first line of defence in the body against PH change. They are present in ECF, ICF, and in the bones. They tend to balance the H+ ions but do not excrete it.

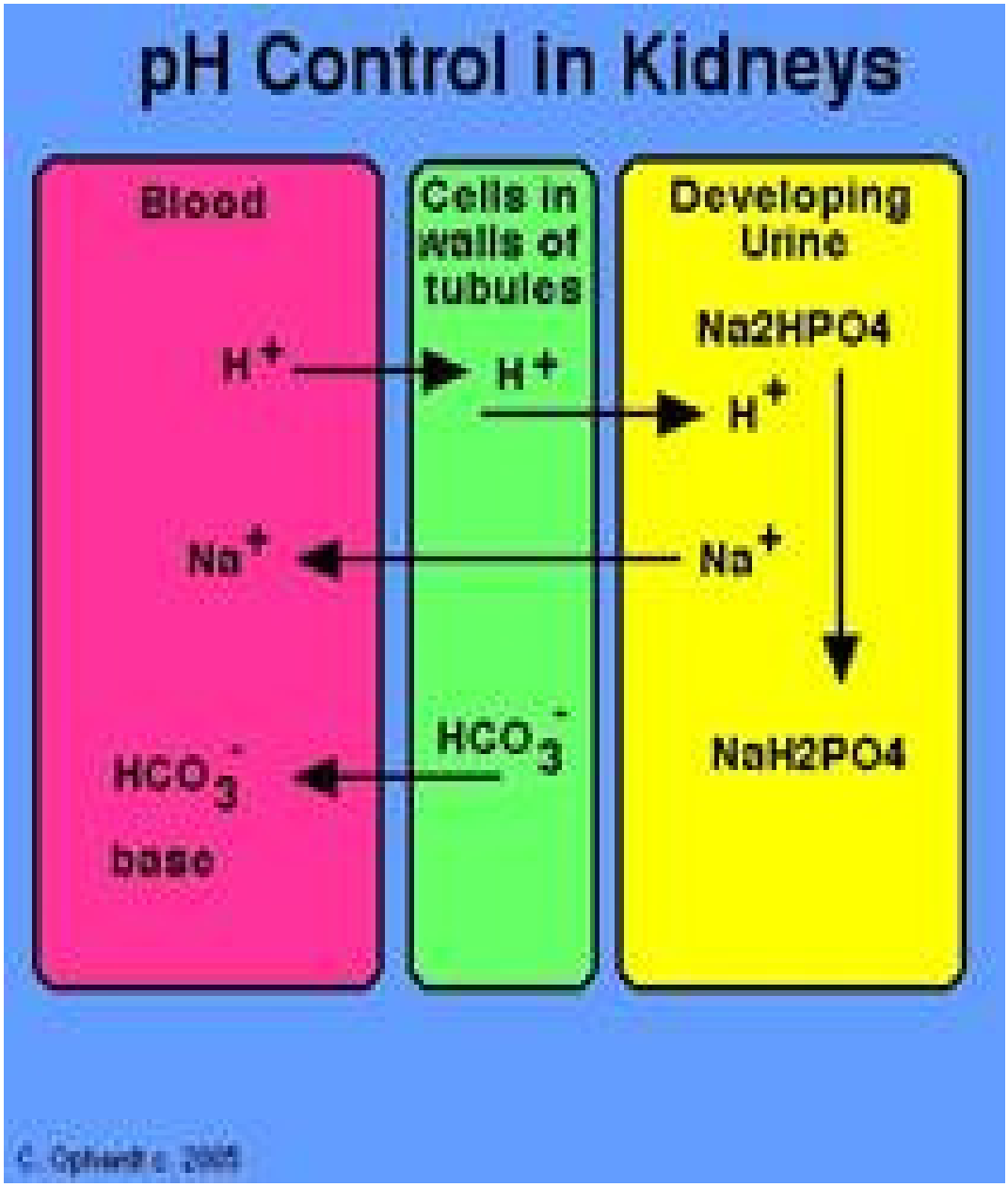
Respiratory response.

These are the second line of defence against PH changes.CO2 is removed through the lungs as fast as it produced by the body. When more amount of CO2 is produced lungs tend to excrete it through hyperventilation..

Renal response.

The renal system stands third in body's buffering mechanism. As already seen, the chemical buffers do not excrete acids, the whole responsibility of acid excretion falls on the kidneys. They do it by combining with anions in the body.

They in addition add Bicarbonates to the ECF to compensate for the acidosis. But it is slower system and full renal compensation may take 1 to 3 days.


pH Control in Kidneys
Blood
Cells in walls of tubules
Developing Urine
Na2HPO4
H+
H+
H+
Na+
Na+
HCO3-
HCO3-
base
NaH2PO4

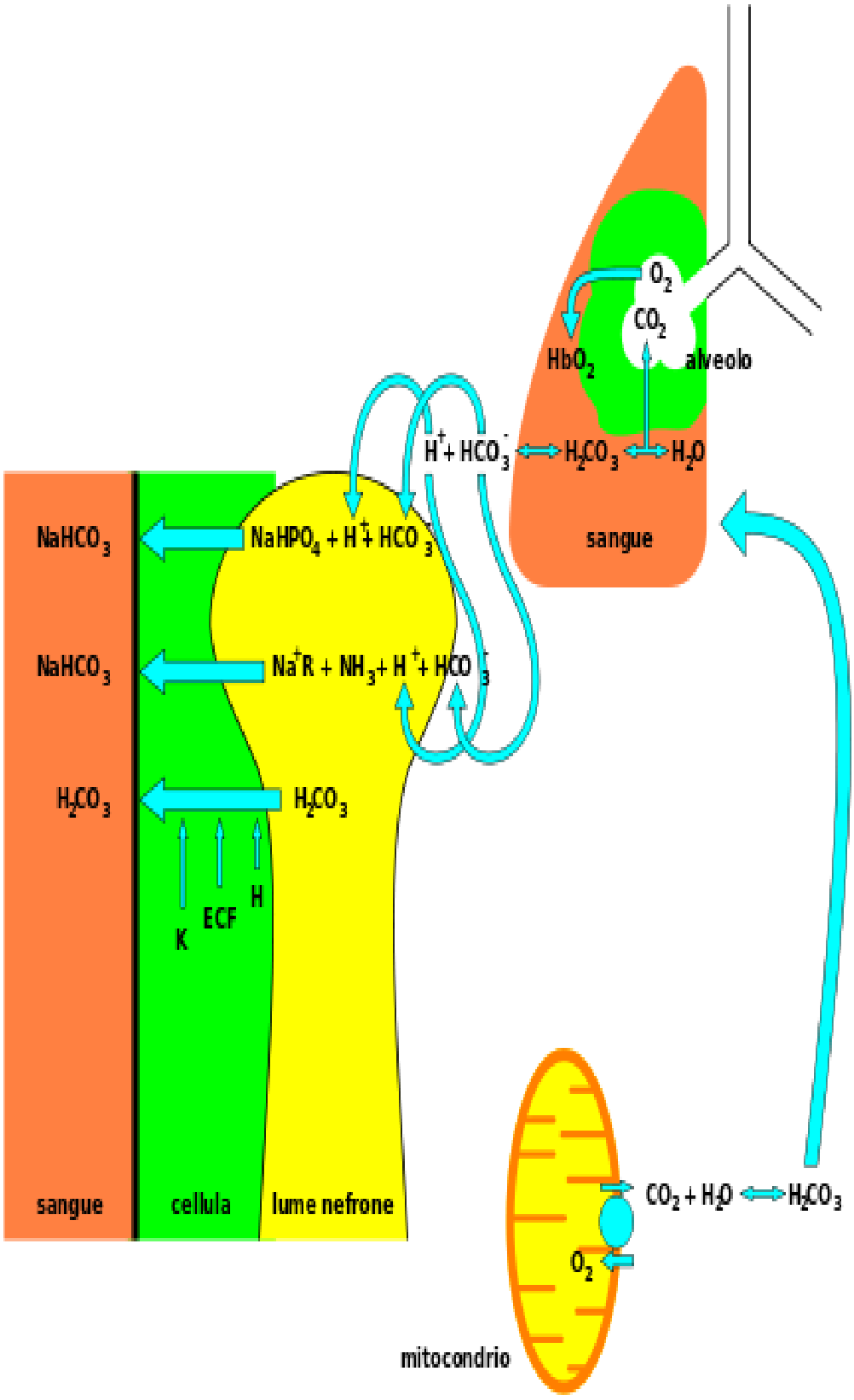
Chemical buffers are the first line of defense against changes in pH.

Major Chemical pH Buffers in the Body.

The body contains many conjugate acid–base pairs that act as chemical buffers . In the ECF, the main chemical buffer pair is HCO_3^-/CO_2. Plasma proteins and inorganic phosphate are also ECF buffers. Cells have large buffer stores, particularly proteins and organic phosphate compounds. $HCO_3^-$is present in cells, although at a lower concentration than in ECF. Bone contains large buffer stores, specifically salts of phosphate and carbonate. When a base or an acid is added to the system the body responds by either adding H+ to the system or removes excess of it . Buffering in ECF occurs rapidly, in minutes.

Bicarbonate/carbondioxide buffer

For several reasons, the HCO_3^-/CO_2 buffer pair is especially important in acid–base physiology: Its components are abundant; the concentration of HCO_3^- in plasma or ECF normally averages 24mmol/L. Although the concentration of dissolved CO_2 is lower (1.2 mmol/L), metabolism provides a nearly limitless supply. It is controlled by the lungs and kidneys.CO_2 exists in the body in several different forms: as gaseous CO_2 in the lung alveoli and as dissolved CO_2, H_2CO_3, HCO_3^-, carbonate (CO_3^{2-}), and carbamino compounds in the body fluids. CO_3^{2-} is present at appreciable concentrations only in alkaline solutions, and so we will ignore it. We will also ignore any CO_2that is bound to proteins in the carbamino form. The most important forms are gaseous CO_2, dissolved CO_2, H_2CO_3, and HCO_3^-.


O_2
CO_2
HbO_2
alveolo
$H^+ + HCO_3^- \leftrightarrow H_2CO_3 \leftrightarrow H_2O$
sangue
$NaHCO_3$
$NaHPO_4 + H^+ + HCO_3^-$
$NaHCO_3$
$Na^+R + NH_3 + H^+ + HCO_3^-$
H_2CO_3
H_2CO_3
K
ECF
H
sangue
cellula
lume nefrone
$CO_2 + H_2O \leftrightarrow H_2CO_3$
O_2
mitocondrio

In th pulmonary system, $CO_{2(d)} = 0.03 \times PCO_2$. If PCO_2 is 40 mm Hg, then $CO_{2(d)}$ is 1.2 mmol/L.

In aqueous solutions, $CO_{2\,(d)} + H_2O \rightleftharpoons H_2CO_3$. This reaction forming

Bicarbonate that is at the right is called hydration reaction while the left reaction is called the dehydration reaction. This reaction is slow and hence catalysed by carbonic anhydrase enzyme. At equilibrium, $CO_{2(d)}$ is greatly favoured; at body temperature, the ratio of $CO_{2(d)}$ to H_2CO_3 is about 400:1. If $CO_{2(d)}$ is 1.2 mmol/L, then H_2CO_3 equals 3 μmol/L. H_2CO_3 dissociates instantaneously into H^+ and HCO_3^-: $H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$.

Bicarbonate/carbon dioxide open system.

As noted previously, the pK of the HCO_3^-/CO_2 system (6.10) is far from 7.40, the normal pH of arterial blood. From this, one might view this buffer pair as rather poor.

How the kidneys and respiratory system influence ECF pH by operating on the HCO_3^-/CO_2 system is described below. For now, the advantages of an open buffer system are best explained by changes in PCO2 and PH values. Another way in which blood pH may be protected is by changes in endogenous acid production . An increase in blood pH caused by the addition of base to the body stimulates production of lactic acid and ketone body acids, which then reduces the alkaline shift in pH. A decrease in blood pH inhibits production of lactic acid and ketone body acids, which diminishes the acidic shift in pH.

This scenario is especially important when the endogenous production of these acids is high, as occurs during strenuous exercise or other conditions of circulatory inadequacy (lactic acidosis) or during ketosis resulting from uncontrolled diabetes, starvation, or alcoholism. These effects of pH on endogenous acid production result from changes in enzyme activities brought about by the pH changes, and they are part of a negative feedback mechanism regulating blood pH.

Lungs are the second line of defense against changes in pH.

Reflex changes in ventilation help to defend blood pH. By changing the PCO_2, and hence H_2CO_3 of the blood, the respiratory system can rapidly and profoundly affect blood pH. A fall in blood pH stimulates ventilation, primarily by acting on peripheral chemoreceptors. An elevated arterial blood PCO_2 is a powerful stimulus to increase ventilation; it acts on both peripheral and central chemoreceptors but primarily on the latter. When CO_2 accumulates in the body, ventilation is stimulated, the lungs blow off more CO_2, thereby making the blood less acidic.

Role of lungs in Acid Excretion.

The lungs senses the change in PH through either Hypercarbia and hypoxia. The excess CO2 in hypercarbia enters causes increased production of H+ ions which stimulates the chemoreceptors in the brain stimulating the respiratory centre inducing hyperventilation. Reverse occurs in hypocarbia and CO2 is retained for normal respiratory drive.

RESPIRATORY ACIDOSIS: increase in co2 decreases the HCO3 /PCO2 ratio. Dissociation carbonic acid produces increase in HCO3- ion so if stil persists kidneys starts conserving HCO3- ion. This is compensated respiratory acidosis. Renal compensation is determined by base deficit.

Increase in co2 is seen in hypoventilation and v/q mismatch.

RESPIRATORY ALKALOSIS:decrease in CO2 concentration increases the HCO_3 /PCO_2 ratio.kidneys start excreting the bicarbonate ion if this persists to maintain the normal ratio. There will be a negative base excess/ base deficit.

Decrease in PCO2 seen in hyperventilation, high altitude

ANAESTHETIC SIGNIFICANCE OF OXYGEN TRANSPORT.

Effects of anaesthesia on gas exchange.

Functional residual capacity (FRC)

It is the volume if air that is normally present in the lungs after normal expiration of tidal volume.

It is nothing but the sum of residual volume which is the air left in the lungs after forced expiration and the expiratory reserve volume which is the air that can be expelled only by forced expiration. Preoxygenation is the process of filling this FRC with Oxygen so that it serves as a reserve for oxygen when the patient is apneic.For filling a FRC of 3000ml with room air with a FiO2 of 21%, 3mins preoxygenation gives 630 ml. So preoxygenation with 100% oxygen takes 3mins to fill FRC with O2.

FRC also changes with posture. Supine reduces the due to the compression of the base of the lungs. General anaesthesia relaxes the diaphragm and the intercostal muscles giving way for the abdominal contents to push the lungs cephalad reducing the FRC still about 20%.It is also reduced by obesity. pregnancy, abdominal distension, etc. So obese patient lying supine reduces his FRC to 50% making him more vulnerable to hypoxia than others due to reduced FRC.

Closing capacity (CC)

It is the volume air at which the smaller airways tend to close causing alveoiar collapse. Normally FRC will be more than Closing capacity and it tends to prevent airway collapse.In general anaesthesia, this CC approaches close to FRC leading to basal atelectasis.This is more in cases of neonates ,smokers,elderly,and patients with respiratory diseases.Experiments found that is seen in about 80% of the patients.

REVIEW OF LITERATURE

A study by Lundborg in 1970 showed experimentally that the intraneuronal microcirculation in the limbs is completely reinstituted after 6-8 hours of ischemia.Thus the nerves in the limbs seems to have a wide margin of safety- Ocha et al 1972.

Mullick 1978 and wilgis 1971 also proved that the cellular metabolic changes during clinical ischemia have been well established by studying venous blood parameters .

Anderson et al 1979 from his studies on reconstructive arterial surgery and tourniquet ischemia showed that there is an increased plasma potassium level after ischemia.

Schmitt *et al*. studied the effects of Fentanyl and midazolam on tourniquet application. Midazolam was administered before tourniquet inflation intravenously. It was seen that there was a sustained increase in serum midazolam after tourniquet release which is more pronounced in patients more than 70 years of age. It is significant in that the effect of the drugs can prolong into the post operative period exerting its effect seriously .

Wiglis studied the duration and its impact. He demonstrated a time dependent increase in acidosis in the venous blood of the limb under tourniquet. He also emphasised that duration of more than 1.5 to 2.0 hours showed ultra-structural changes in the muscle fibres.

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Newman in his study has found out that most of the muscle ATP, and glycogen stores are depleted when the tourniquet duration exceeds 1.5to2 hours.

The recovery period after deflation mainly depends on the replenishment of the ATP stores after removal of tourniquet.

John kendrew who was awarded the nobel prize for his work on myoglobin found out that mice who were genetically engineered showed a lack of this myoglobin pigment and they also showed a reduction in the stroke volume of about 30% due to impaired myocardial contractility probably due to their defeciency of myoglobin.He has found out that these mice adapted to this defeciency by vasodilatation and reflex reactions to hypoxia.

MATERIALS AND METHODS

STUDY TYPE : INTERVENTIONAL.

DESIGN OF STUDY : PROSPECTIVE RANDOMISED CASE CONTROL STUDY

SELECTION OF STUDY SUBJECTS : CASES POSTED FOR LIMB SURGERIES

DATA COLLECTION : DATA REGARDING HISTORY, CLINICAL EXAMINATION, RADIOLOGICAL EXAMINATION.

INCLUSION CRITERIA:

Elective limb surgeries.

Both sexes

Age: 18-65 years

ASA I & II

HB more than 10.0gms.

EXCLUSION CRITERIA:

Patient's refusal.

Patients with documented neuromuscular disorders.

Patients with respiratory compromise.

Patients of cardiovascular disease.

Patients with renal disorder.

METHODOLOGY

Patients scheduled for limb surgeries using tourniquet are eligible for the study. 60 patients are randomized into two groups. In a randomized manner 30 patients received nasal oxygen 3 l/min after tourniquet application (Group 1), and 30 patients receive preoxygenation for 5mins before tourniquet in addition to 3 l/min nasal oxygen during tourniquet (Group 2).Arterial blood samples and venous blood samples are collected before and 3mins after tourniquet respectively and analysed for blood gases and lactate levels.

Statistical Tools.

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square, ‘t’ value and 'p' values were calculated. ‘t’ test was used to test the significance of difference between quantitative variables and Yate’s and Fisher’s chi square tests for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship

PARAMETERS TO BE MONITORED:

PH,Hco3,Lactate,Pco2,Pao2.

PR, BP, SPO2,PCO2.

OBSERVATIONS AND RESULTS.

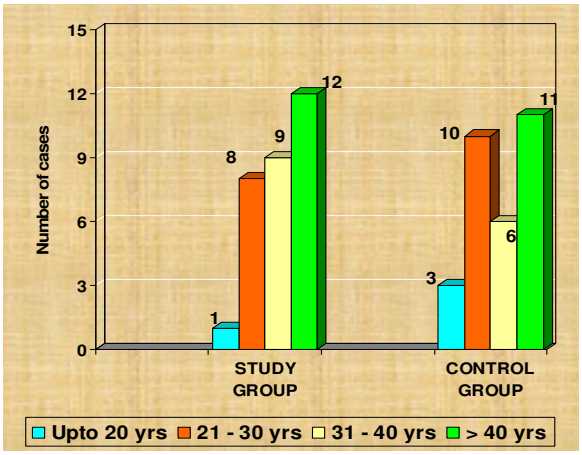
A:PROFILE OF CASES STUDIED

Table A1: Age Distribution

Age Group	No of Cases in			
	Study Cases		Controls	
	No	%	No	%
Up to 20 yrs	1	3.3	3	10
21 – 30 yrs	8	26.7	10	33.3
31 – 40 yrs	9	30	6	20
Above 40 yrs	12	40	11	36.7
Total	30	100	30	100
Range	19 – 46 yrs		18 – 45 yrs	
Mean	35.8 yrs		33.7 yrs	
SD	8.1 yrs		9.4 yrs	
'p'	**0.3497 Not significant**			

As indicated in the above table the age characteristics of both the case and control group are compared and there are similar and no statistical difference exists between the two.

AGE DISTRIBUTION



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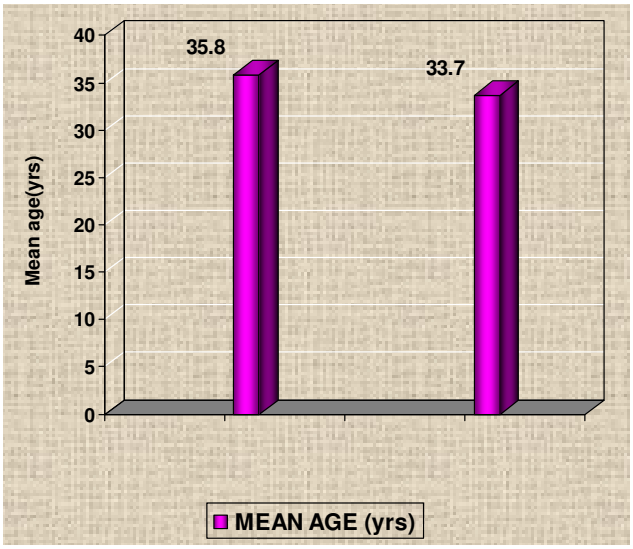
MEAN AGE



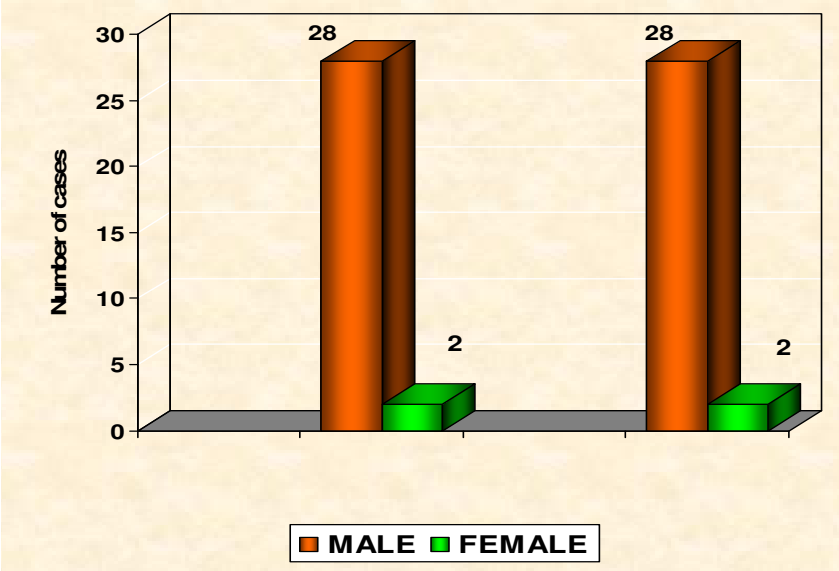
As indicated above the mean age of both the group are not statically significant.

Table A2: Sex Distribution

Sex	No of Cases in			
	Study Group		Control Group	
	No	%	No	%
Male	28	93.3	28	93.3
Female	2	6.7	2	6.7
'p'	1.0 Not significant			

There is no statistical difference between the sex distribution of both the groups.

SEX DISTRIBUTION



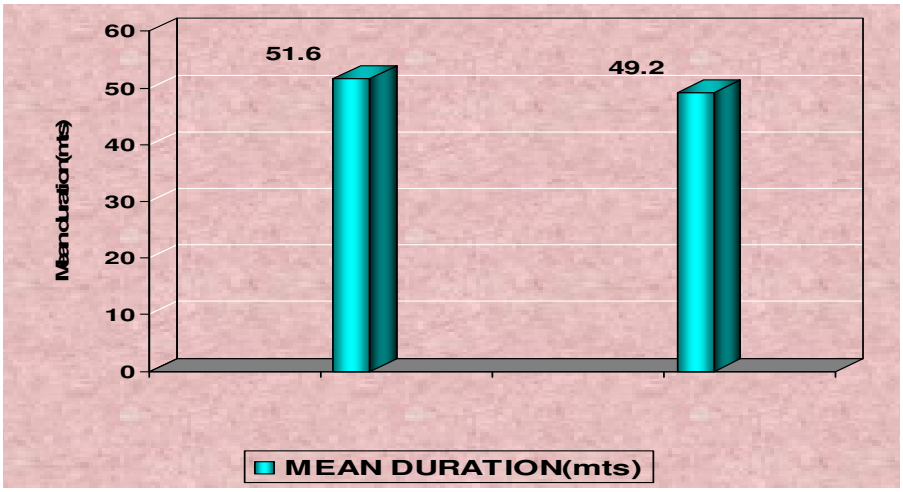
There is no statistical difference between the sex distribution of both the groups.

Table A3: Duration

Group	Duration of procedure (in minutes)	
	Mean	**SD**
Study	51.6	7.5
Controls	49.2	7.7
'p'	0.2199 Not significant	

As indicated above there is no statistical difference between the duration in both the groups.

DURATION



As indicated above there is no statistical difference between the duration in both the groups.

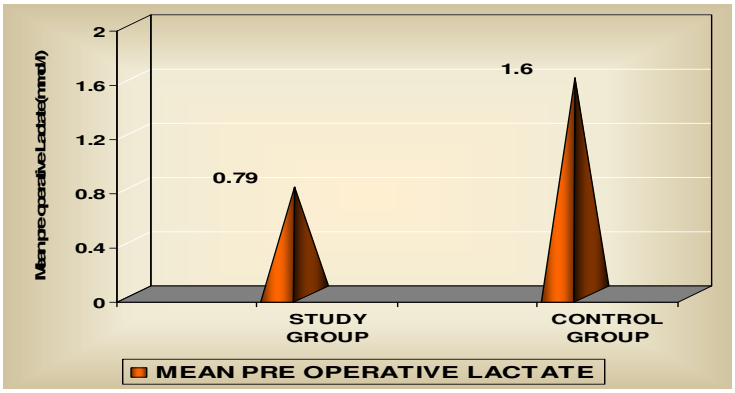
TABLE B: OPERATIVE VALUES

Table B1: Lactate (mmol/l)

Group	**Lactate (mmol/l)**	
	Mean	**SD**
Study	0.79	0.18
Controls	1.6	0.48
'p'	**<0.0.0001 Significant**	

As evidenced by the above table there is statistical difference between the lactate levels in both the groups indicating a higher lactate level in control group.

LACTATE



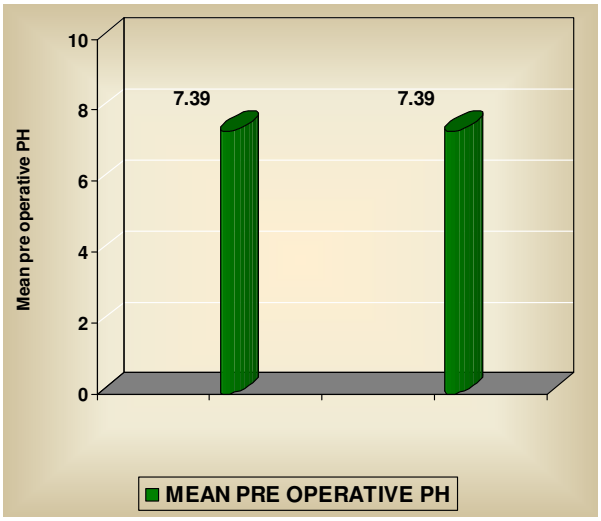
As evidenced by the above table there is statistical difference between the lactate levels in both the groups indicating a higher lactate level in control group.

Table B2: Preoperative PH Value

Group	Pre operative PH	
	Mean	**SD**
Study	7.39	0.02
Controls	7.39	0.02
'p'	0.351 Not significant	

As seen above there is no statistical significant difference between the Pre operative PH values of both groups.

PRE OPERATIVE PH



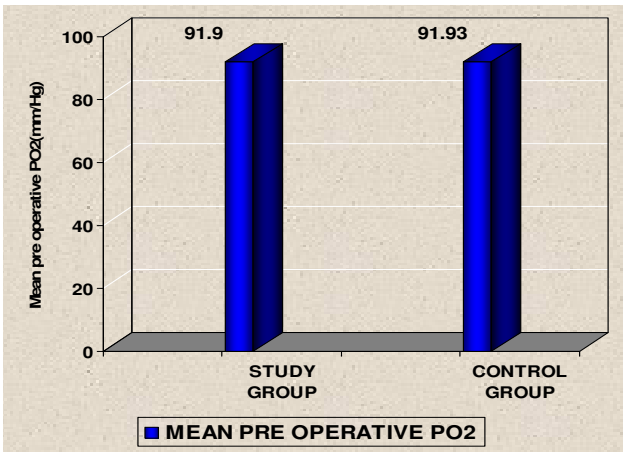
As seen above there is no statistical significant difference between the Pre operative PH values of both groups.

Table B3: Preoperative PO_2 (mm/Hg)

Group	Pre operative PO2 (mm/Hg)	
	Mean	**SD**
Study	91.9	1.88
Controls	91.93	2.08
'p'	0.9484 Not significant	

There is no statistical difference between the preoperative PO2 values in both the groups.

PRE OPERATIVE PO2



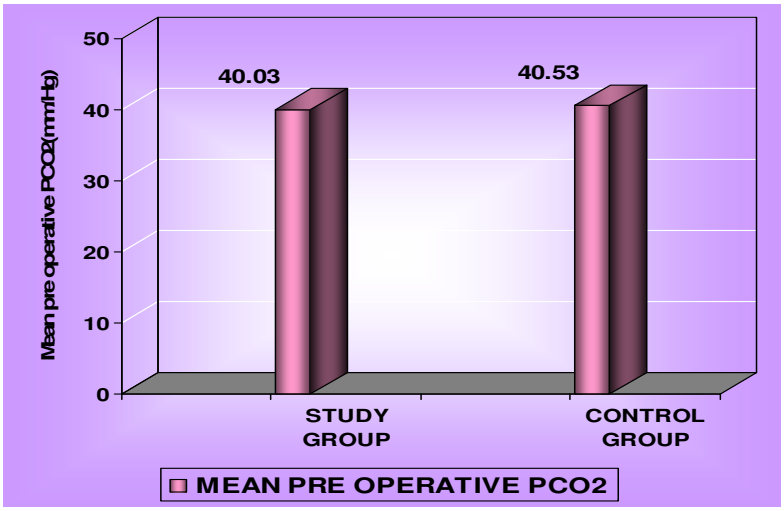
There is no statistical difference between the preoperative PO2 values in both the groups.

Table B4:Pre operative PCO2 (mm/Hg)

Group	Pre operative PCO2 (mm/Hg)	
	Mean	**SD**
Study	40.03	1.97
Controls	40.53	2.18
'p'	0.3552 Not significant	

There is no statistical significant difference between the preoperative PCO2 values in both the groups.

PRE OPERATIVE PCO2



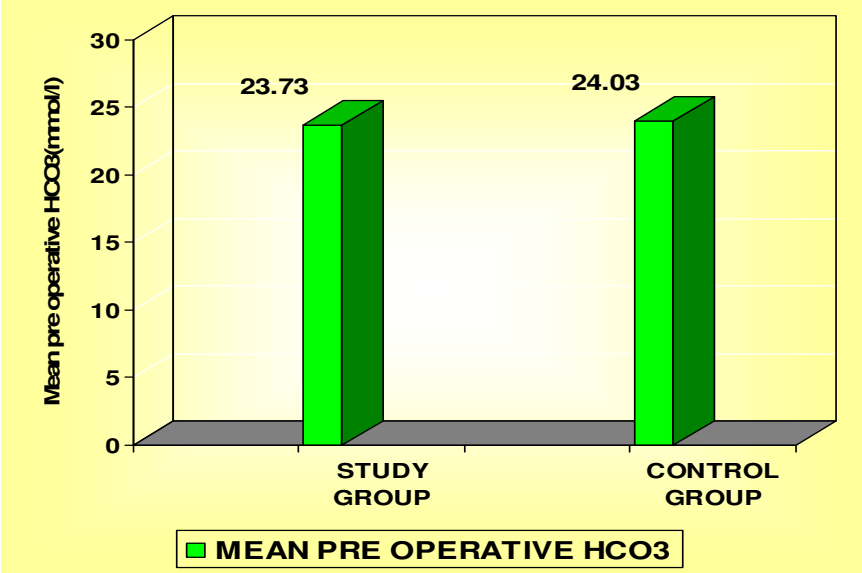
There is no statistical significant difference between the preoperative PCO2 values in both the groups.

Table B5: Pre operative HCO3

Group	**Pre operative HCO3 (mmol/l)**	
	Mean	**SD**
Study	23.73	1.23
Controls	24.03	1.4
'p'	0.3818 Not significant	

There is no statistical difference between the preoperative bicarbonate levels in both the groups.

PRE OPERATIVE HCO3



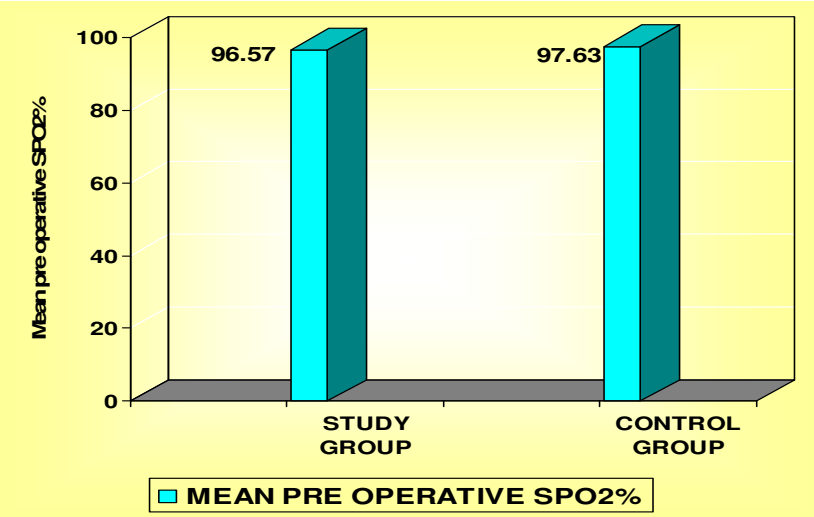
There is no statistical difference between the preoperative bicarbonate levels in both the groups.

Table B6: Pre operative SPO2 (%)

Group	Pre operative SPO2 (%)	
	Mean	**SD**
Study	96.57	1.72
Controls	97.63	1.4
'p'	**0.3107 Not Significant**	

There is no statistical significant difference between the preoperative SPO2 values in both the groups.

PRE OPERATIVE SPO2%



There is no statistical significant difference between the preoperative SPO2 values in both the groups.

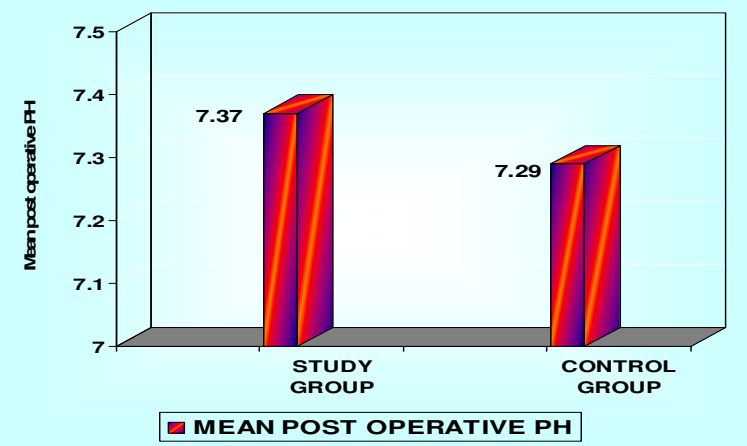
C: POST OPERATIVE VALUES

Table C1: Postoperative PH

Group	Post operative PH	
	Mean	**SD**
Study	7.37	0.03
Controls	7.29	0.01
'p'	**<0.0001 Significant**	

There is a statistical significant difference between both the groups in terms of PH with control group showing more acidosis.

POST OPERATIVE PH



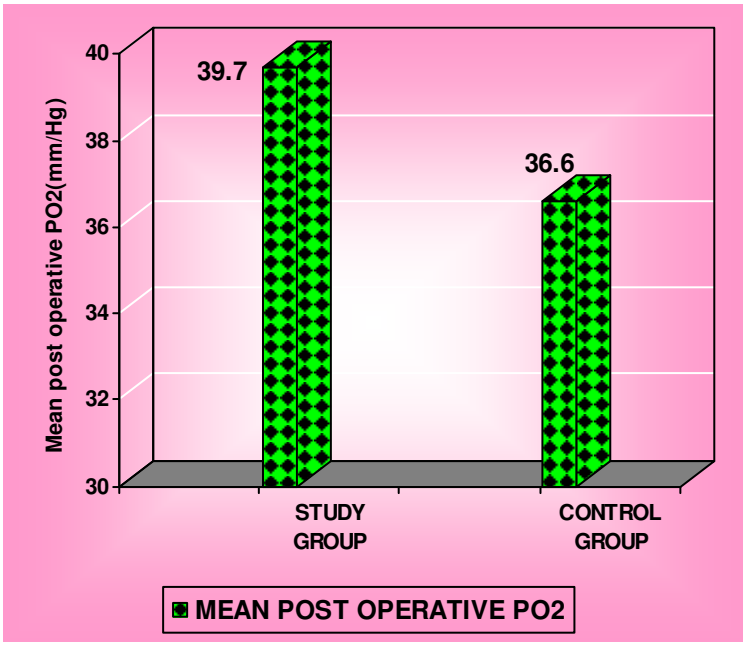
There is a statistical significant difference between both the groups in terms of PH with control group showing more acidosis.

Table C2: Postoperative PO2 (mm/hg)

Group	Post operative PO2 (mm/hg)	
	Mean	**SD**
Study	39.7	1.53
Controls	36.6	1.77
'p'	**<0.0001 Significant**	

There is a statistical significant difference between both the groups in post operative PO2 values.

POST OPERATIVE PO2



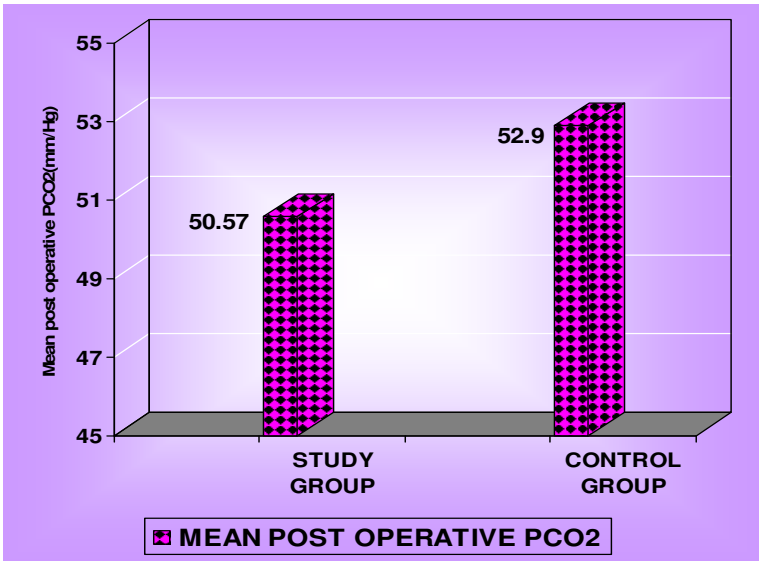
There is a statistical significant difference between both the groups in post operative PO2 values.

Table C3: Post operative PCO2 (mm/Hg)

Group	Post operative PCO2 (mm/Hg)	
	Mean	**SD**
Study	50.57	2.61
Controls	52.9	1.79
'p'	**0.0002 Significant**	

There is a statistical significant difference between the post operative PCO2 values in both the groups.

POST OPERATIVE PCO2



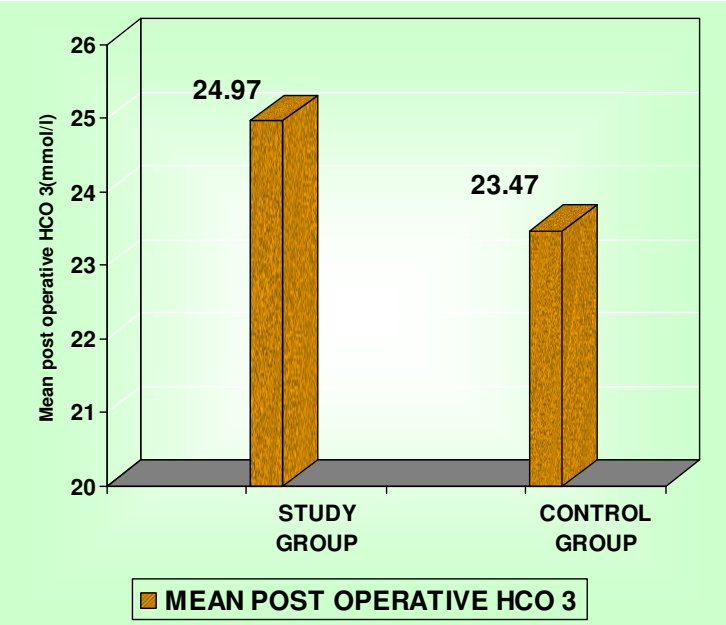
There is a statistical significant difference between the post operative PCO2 values in both the groups.

Table C4: Postoperative HCO3

Group	Post operative HCO3 (mmol/l)	
	Mean	**SD**
Study	24.97	1.63
Controls	23.47	1.2
'p'	**0.0001 Significant**	

There is a statistical significant difference between the post operative HCO3 values in both the groups.

POST OPERATIVE HCO 3



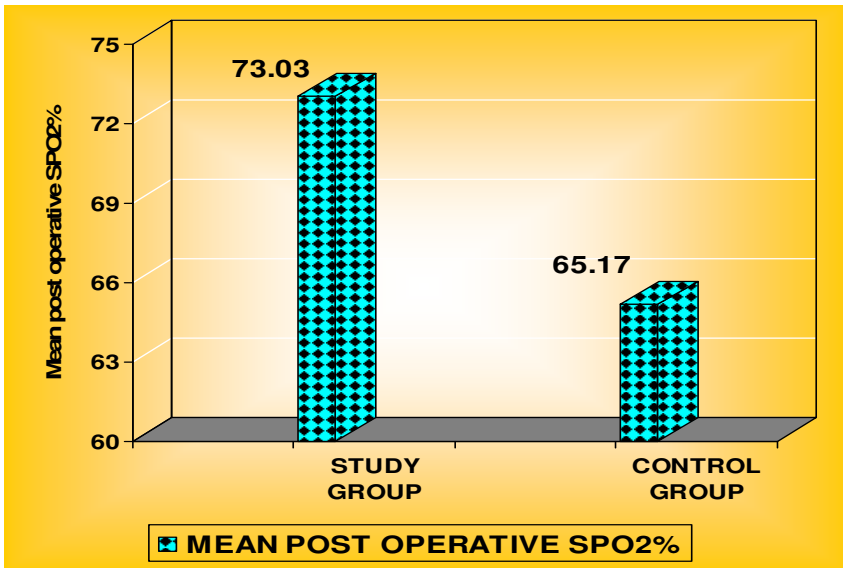
There is a statistical significant difference between the post operative HCO3 values in both the groups.

Table C5: Postoperative SPO2

Group	Postoperative SPO2 (%)	
	Mean	**SD**
Study	73.03	4.31
Controls	65.17	2.56
'p'	**<0.0001 Significant**	

There is a statistical significant difference between the post operative SPO2values in both the groups.

POST OPERATIVE SPO2 %



There is a statistical significant difference between the post operative SPO2values in both the groups.

DISCUSSION

Tourniquet application is one of the preferred methods for obtaining a bloodless surgical field. At the same time it has its own disadvantages. It has the tendency to increase the anaerobic metabolism due to oxygen deprivation in the tissues under touniquet. This study was done in a trial to reduce the anaerobic metabolism.

Newman in his study has found that when the duration of tourniquet exceeds 30mins, anaerobic metabolism set in. Similar to his study, our study also shows anaerobic metabolism as evidenced by increase in lactate levels since the average duration of tourniquet in our study is about 45mins.

In this study, the Age and sex characteristics of both the groups were compared and found to be not statistically significant.

The mean pre operative PH of both the case and control group was 7.39±0.02 and that was not statistically significant.

The mean post operative PH of study group was 7.37±0.03 and that of the control group was 7.29±0.01.These values were found to be statistically significant.

The mean Serum lactate levels in the study group was 0.79 with a standard deviation of 0.18 while that of the control group was 1.6±0.48.These were also found to be statistically significant.

By supplementing oxygen, we can improve the oxygen content of blood thereby increasing the reserve oxygen. This increased reserve oxygen can help in the period of oxygen deficiency during tourniquet. The supplemental oxygen as given in the form of preoxygenation increases the fraction of dissolved oxygen in the blood there by increasing the oxygen reserve. This reserve is the key for the favorable outcome.

However this study has its own limitations. The tourniquet pressure was not standardised as some surgeries are done using pneumatic tourniquet while the others using compression esmark tourniquet. Moreover, CPK, serum Lactic acid levels, and quantitative H+ concentrations could not be done due to the lack of facilities. Even if they were done they have the impact of the primary pathology rather than the tourniquet alone.

However this study could be done in more sample of patients with more relevant and added study parameters in future to increase its practical significance.

SUMMARY

In this study we evaluated the effect of oxygen supplementation in tourniquet used limb surgeries. Sixty patients of ASA 1and 2 were divided into two groups of 30 each.

GROUP 1: Receive nasal oxygen 3 l/min throughout the surgery.

GROUP 2: Preoxygenated for 5mins before tourniquet application and receive oxygen 3 l/min throughout the procedure.

The study was conducted in Govt.Rajaji Hospital and Madurai Medical College Madurai, from September 2013 to August 2014.

In this study, the Age and sex characteristics of both the groups were compared and found to be not statistically significant.

Similarly, the Preoperative PH, PO_2, PCO_2 of both the groups were found to be not statistically significant.

The preoperative values of HCO_3, SPO_2 were compared in both the groups and found to be statistically not significant.

The mean duration of tourniquet application in both the groups were similar and found to be statistically not significant.

So both the groups were similar in the study parameters before tourniquet application.

When both the groups were compared with respect to their post tourniquet values, it was found that the group which received preoxygenation showed reduced anaerobic metabolites and reduced serum lactate when

compared to the group which was not preoxygenated. This difference in serum lactate was found to be statistically significant.

Similarly the PH of the group which was preoxygenated was found within the normal limit while the group not preoxygenated exibited an acidotic PH.This difference in PH was also found to be statistically significant.

CONCLUSION

The effect of Oxygen supplementation in reducing the anaerobic metabolism was studied in patients undergoing limb surgeries using tourniquet.

It was found that the patients who received preoxygenation showed reduced serum Lactate levels and normal PH when compared to those who were not preoxygenated.

Hence it is concluded that Preoxygenation reduces anaerobic metabolism in surgeries done under tourniquet.

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PROFORMA

NAME : I.P.NO: ASA:

AGE & SEX:

WEIGHT :

DATE& TIME OF ADMISSION:

DIAGNOSIS:

PROCEDURE:

HISTORY: NEUROMUSCULAR DISORDER

CVS PATHOLOGY

RENAL DISEASE

PULMONARY PATHOLOGY.

CLINICAL EXAMINATION:

PR,BP, SPO2, RS, CVS

BASIC INVESTIGATIONS:

HAEMOGLOBIN

RENAL PARAMETERS &SERUM ELECTROLYTES,

CHEST X RAY PA VIEW

ECG.

ANAESTHETIC TECHNIQUE:

Routine spinal anaesthesia for lower limb surgeries and Supraclavicular block for upper limb surgeries and tourniquet application.

GROUP 1: Receive nasal oxygen 3 l/min throughout the surgery.

GROUP2: Preoxygenated for 5 mins before tourniquet application and receive oxygen 3 l/min throughout the procedure.

TOURNIQUET DURATION:

TOURNIQUET PRESSURE:

DURATION OF SURGERY: Hrs.

TEMPERATURE:

ABG	PH	PaO2	Pco2	HCO3	LACTATE	H+	MAP	PR	SPO2
PRE TOURNIQUET									
POST TOURNIQUET									

COMPLICATIONS IF ANY:

MASTER CHARTS - CASES

											Preop					POSTOP				
Name	Age	Sex	IP no	ASA	HB	Duration	CVS	RS	RFT	Lactate	PH	PO2	PCO2	HCO3	SPO2	PH	PO2	PCO2	HCO3	SPO2
athul	28	M	1232	1	10.2.	58	N	N	N	0.6	7.36	93	37	24	97	7.37	39	50	27	66
bhuban	34	M	56743	1	10.5	55	N	N	N	0.8	7.38	90	38	22	98	7.33	38	52	23	78
vulavn	19	M	65478	1	10.8	48	N	N	N	0.5	7.41	93	39	23	99	7.4	37	53	24	69
rajesh	43	M	89009	1	11	45	N	N	N	0.9	7.39	94	42	23	95	7.37	41	46	24	72
alagar	46	M	56743	2	11.8	56	N	N	N	1	7.4	89	39	25	96	7.34	40	45	27	77
muthu	39	M	44567	1	12.2	60	N	N	N	0.6	7.37	93	41	26	99	7.32	39	44	27	79
pathy	42	M	65212	2	10.3	40	N	N	N	0.8	7.37	91	43	23	96	7.41	38	48	24	68
nambi	29	M	34212	1	10.4	45	N	N	N	0.7	7.39	95	42	22	100	7.4	39	49	23	70
umar	24	M	34532	1	10.9	62	N	N	N	1.1	7.42	93	40	25	98	7.32	40	47	27	74
ruthran	31	M	45212	1	10	49	N	N	N	0.8	7.4	90	42	24	97	7.33	41	51	24	75
mani	45	M	44322	1	11	58	N	N	N	0.6	7.37	93	41	26	95	7.38	42	52	28	79
josnem	46	M	32111	1	11.5	55	N	N	N	0.8	7.37	91	43	23	95	7.39	40	50	24	68
ponnu	42	F	34567	1	12.2	48	N	N	N	0.5	7.39	95	42	22	97	7.33	38	48	23	70
minath	39	M	89009	1	10.5	45	N	N	N	0.9	7.42	93	40	25	95	7.36	39	49	27	74
thiyagu	26	M	45671	2	10.4	56	N	N	N	1	7.4	90	42	24	94	7.35	40	53	24	75
durai	37	M	234	1	10.2	60	N	N	N	0.6	7.36	93	37	24	95	7.39	40	51	25	66
krishnan	43	M	8906	1	10.1	40	N	N	N	0.8	7.38	90	38	22	97	7.41	41	51	23	78
mohan	44	M	7854	1	10.8	45	N	N	N	0.7	7.41	93	39	23	96	7.38	43	54	24	69
sankar	37	M	56743	1	11.8	62	N	N	N	1.1	7.39	94	42	23	99	7.38	42	54	24	72
ammu	25	F	345	2	10.4	49	N	N	N	0.8	7.4	89	39	25	95	7.36	40	51	27	77
faroo	21	M	66789	1	10.4	45	N	N	N	0.7	7.42	93	40	25	94	7.39	38	52	26	74
ezhilan	29	M	30091	1	10.3	56	N	N	N	0.9	7.4	90	42	24	96	7.37	39	54	25	75
varman	38	M	45673	2	10.6	60	N	N	N	0.5	7.36	93	37	24	95	7.36	37	51	26	66
bala	41	M	34562	2	11.6	45	N	N	N	0.7	7.42	93	40	25	96	7.38	41	52	27	74
wilson	43	M	34521	1	11.5	56	N	N	N	1	7.4	90	42	24	98	7.41	42	53	24	75
umapathy	44	M	23432	1	12.1	60	N	N	N	0.7	7.36	93	37	24	99	7.4	40	51	25	66
ravi	26	M	23890	1	12.4	40	N	N	N	0.8	7.38	90	38	22	95	7.35	41	50	23	78
sudakar	35	M	45890	1	11.2	62	N	N	N	1	7.39	94	42	23	97	7.43	39	53	24	72
alagar	42	M	23451	1	10.3	49	N	N	N	1.1	7.4	89	39	25	95	7.43	38	51	27	77
pandi	37	M	11134	1	10.4	40	N	N	N	0.8	7.38	90	38	22	99	7.4	39	52	23	78

MASTER CHART - CONTROL

									Preop					Postop					
Name	**Age**	**Sex**	**IP No**	**ASA**	**HB**	**Duration**	**CVS**	**RS**	**RFT**	**PH**	**PO2**	**PCO2**	**HCO3**	**SPO2**	**PH**	**PO2**	**PCO2**	**HCO3**	**SPO2**	**Lactate**
minath	23	M	45671	2	10.4	61	N	N	N	7.38	89	38	23	97	7.28	38	52	23	67	1.5
thiyagu	35	M	234	1	10.2	42	N	N	N	7.41	92	40	24	98	7.29	34	53	24	68	1.8
durai	43	M	8906	1	10.1	40	N	N	N	7.39	93	42	25	99	7.3	35	55	24	60	2
krishnan	44	M	7854	1	10.8	61	N	N	N	7.4	95	43	23	97	7.28	35	51	23	64	1.7
mohan	21	M	56743	1	11.8	45	N	N	N	7.37	91	41	24	95	7.27	38	54	25	65	1
sankar	19	M	345	2	10.4	50	N	N	N	7.37	93	42	26	96	7.3	34	55	22	66	0.7
sakthi	35	M	66789	1	10.4	52	N	N	N	7.39	94	39	25	95	7.29	39	50	25	64	2.2
faroo	26	M	30091	1	10.3	44	N	N	N	7.38	95	38	22	98	7.3	37	51	25	65	1.9
ezhilan	39	M	45673	2	10.6	40	N	N	N	7.4	92	40	24	99	7.28	37	54	24	63	1.1
varman	38	M	34562	2	11.6	50	N	N	N	7.43	91	42	23	98	7.3	38	55	23	62	2.4
bala	41	M	34521	1	11.5	50	N	N	N	7.37	89	41	26	98	7.3	34	55	24	63	1.8
wilson	45	M	23432	1	12.1	52	N	N	N	7.39	95	43	25	97	7.29	39	50	22	66	1.9
umapathy	27	M	23890	1	12.4	44	N	N	N	7.42	92	43	24	97	7.3	37	51	23	67	2
ravi	45	M	45890	1	11.2	40	N	N	N	7.4	93	40	23	100	7.28	37	54	24	68	1.2
sudakar	21	M	23451	1	10.3	50	N	N	N	7.43	94	39	25	96	7.3	38	55	24	69	1
alagar	28	M	11134	1	10.4	61	N	N	N	7.38	90	37	26	97	7.28	38	52	24	65	0.9
pandi	25	M	1232	1	10.2	42	N	N	N	7.41	91	38	24	98	7.29	34	53	23	63	2.2
chinnapon	18	F	56743	1	10.5	40	N	N	N	7.39	89	39	22	98	7.3	35	55	22	64	1.2
vulavn	29	M	65478	1	10.8	61	N	N	N	7.4	93	36	23	96	7.28	35	51	21	63	1.1
rajesh	37	M	89009	1	11	45	N	N	N	7.37	95	45	22	100	7.27	38	54	25	70	2.2
alagar	42	M	56743	2	11.8	61	N	N	N	7.38	90	44	24	97	7.28	38	52	23	63	1.9
muthu	41	M	44567	1	12.2	61	N	N	N	7.4	89	42	26	98	7.28	35	51	26	62	1.6
puspam	28	F	65212	2	10.3	45	N	N	N	7.37	94	40	25	96	7.27	38	54	23	70	2.1
nambi	19	M	34212	1	10.4	50	N	N	N	7.37	91	41	25	100	7.3	34	55	24	63	1.2
umar	42	M	34532	1	10.9	52	N	N	N	7.39	92	42	24	97	7.29	39	50	21	68	1.6
ruthran	44	M	45212	1	10	44	N	N	N	7.42	91	41	26	98	7.3	37	51	22	68	0.9
mani	45	M	44322	1	11	40	N	N	N	7.4	93	43	21	99	7.28	37	54	23	65	2
josnem	39	M	32111	1	11.5	50	N	N	N	7.43	94	40	22	97	7.3	38	55	24	64	1.2
ponnu	27	M	34567	1	12.2	61	N	N	N	7.38	90	38	25	98	7.28	38	52	24	63	1.6
malan	45	M	89009	1	10.5	42	N	N	N	7.41	88	39	24	100	7.29	34	53	24	67	2

TO STUDY THE EFFECT OF OXGEN

SUPPLEMENTATION IN TOURNIQUET USED LIMB

SURGERIES BY USING BLOOD GAS ANALYSIS

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

APRIL 2015

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

TAMILNADU

The following project was approved

Name of the PG Student	Course	Name of the project	Remarks
Dr.R.G.Sivabalan, sivabalanrg04@rediffmail.com	**PG in MD (Anesthesiology), Madurai Medical College and Government Rajaji Hospital, Madurai**	**To study the effect of oxygen supple-mentation in tourniquet used limb surgeries by using blood gas analysis.**	**Approved**

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.

2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the E thical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary
Ethical Committee

Secretary
Ethical committee

Chairman

25.4.14
DEAN/Convenor
Madurai Medical College & Govt. Rajaji Hospital, Madurai- 20.

To
The above Applicant
-thro. Head of the Department concerned

Forward to Dr. Sivabalan

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
Govt. Rajaji Hospital
Madurai-625 020

Ref.No.3372/E1/5/2014

Madurai Medical College,
Maduai -20. Dated: 04.2014.

Institutional Review Board/Independent Ethics Committee
Capt.Dr.B.Santhakumar,MD (FM). deanmdu@gmail.com
Dean, Madurai Medical College &
Government Rajaji Hospital, Madurai 625 020 . **Convenor**

Sub: Establishment – Madurai Medical College, Madurai-20 –
Ethics Committee Meeting – Meeting Minutes - for April 2014 – Approved list – reg.

The Ethics Committee meeting of the Madurai Medical College, Madurai was held on 25th April 2014 at 10.00 Am to 12.00 Noon at Auditorium Hall at Govt. Rajaji Hospital, Madurai . The following members of the Ethical Committee have attended the meeting.

1.**Dr.V.Nagarajan,M.D.,D.M(Neuro)** Ph: 0452-2629629 Cell No.9843052029 nag9999@gmail.com.	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	**Chairman**
2.**Dr.Mohan Prasad, MS.M.Ch.** Cell.No.9843050822 (Oncology) drbkcmp@gmail.com	Professor & H.O.D of Surgical Oncology (Retired) D.No.32, West Avani Moola Street, Madurai.-1	**Member Secretary**
3.Dr.K.Parameswari, MD(Pharmacology) Cell No.9994026056 drparameswari@yahoo.com.	Director of Pharmacology Madurai Medical College.	**Member**
4.Dr.S.Vadivel Murugan, MD., (Gen.Medicine) Cell No.9566543048 svadivelmurugan_2007@rediffmail.com.	Professor & H.O.D of Medicine Madurai Medical College	**Member**
5. Dr.L.Santhanalakshmi, MD (Physiology) Cell No.9842593412 dr.l.santhanalakshmi@gmail.com.	Vice Principal, Prof. & H.O.D. Institute of Physiology Madurai Medical College	**Member**
6.Dr.A.Sankaramahalingam, MS., (Gen. Surgery) Cell.No.9443367312 chandrahospitalmdu@gmail.com	Professor & H.O.D. Surgery Madurai Medical College. Madurai	**Member**
7.Mrs.Mercy Immaculate Rubalatha, M.A., Med., Cell.No.9367792650 lathadevadoss86@gmail.com	50/5, Corporation Officer's Quarters, Gandhi Museum Road, Thamukam, Madurai-20.	**Member**
8.Thiru.Pala.Ramasamy, B.A.,B.L., Cell.No.9842165127 palaramasamy2011@gmail.com	Advocate, D.No.72,Palam Station Road, Sellur, Madurai-20.	**Member**
9.Thiru.P.K.M.Chelliah, B.A., Cell No.9894349599 pkmandco@gmail.com	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20.	**Member**